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A SYSTEM  
OF  
CLINICAL MEDICINE







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# A SYSTEM OF CLINICAL MEDICINE

DEALING WITH THE  
*DIAGNOSIS, PROGNOSIS, AND TREATMENT*  
OF DISEASE

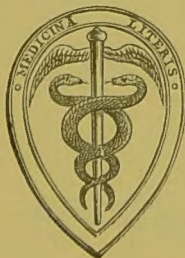
FOR  
STUDENTS AND PRACTITIONERS

BY  
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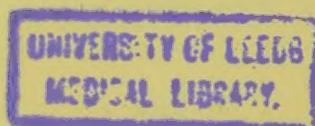
*LOCAL DISEASES AND MICROBIC DISORDERS*



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## PREFATORY NOTE.

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THE amount of matter comprised in the scheme of clinical medicine described in the following pages has necessitated its division into two parts.


The first or larger part deals with all the so-called Local Diseases (diseases causing local symptoms) and the Acute Specific Fevers (microbic disorders).

The second volume, which will appear shortly, will treat of the various conditions attended by Anæmia, Debility without Pyrexia, Symptoms referable to the Extremities, to the Skin, and the Nervous System, and will conclude with a chapter on clinical chemistry, bacteriology, and blood examination.

Each volume will be complete in itself.

THOS. D. SAVILL.

UPPER BERKELEY STREET, LONDON,  
*January, 1903.*



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# INTRODUCTION.

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THOSE who ponder on general principles and methods will have observed that a considerable change has gradually taken place during the last half-century in the methods of EVOLUTION. studying the science and art of medicine. Formerly, men were content to observe the symptoms or effects of disease at the bedside and in the dead-house, and to speculate on the etiological connection of these two series of phenomena. Wherever the association of such phenomena during life and after death was sufficiently constant they were spoken of collectively as a "disease"; when a group of symptoms without anatomical lesion constantly recurred, it received a name and place among the list of "disorders." Then adopting a converse process, each disease or disorder was taken seriatim, its anatomy, symptoms, course, and treatment were described and its various possible etiological factors discussed; and the result was known as "Descriptive" or "Systematic Medicine." It was based very largely, if not solely, on the method of *observation*, and the guiding principle of the process was the tracing from a known effect to an assumed cause.

In later times great advances were achieved, almost synchronously, in two very different directions. On the one hand great improvements were made in the methods of observing and investigating the symptoms or effects of disease during life, and thus Clinical Medicine came into separate existence. This stage was marked by the appearance in this country of two very successful works, one by Dr. Samuel Fenwick, of London, on "Medical Diagnosis," first published in 1869, dealing with the symptoms and diagnosis of disease; another by Dr. James Finlayson, of Glasgow, entitled "A Clinical Manual," first published in 1878, dealing with the methods of observing and

investigating the symptoms of disease. On the other hand, with the extremely rapid growth of chemical, biological, and bacteriological sciences, and the introduction of experimental methods into the investigation of disease processes, a new school of pathology arose, whose methods were based upon *experiment*, and whose leading principle was the artificial production of a cause and the tracing of its effects. The extraordinary advances made by these means, and the new light thus shed upon the science of medicine during the last twenty years, form at once the wonder and delight of the civilised world. Thus, in the present day, we have two schools opposite in their methods and modes of thought, and, to some extent, separate in their aims, the clinical observer on the one hand and the experimental pathologist on the other.

It would be out of place to discuss the relative methods of these schools. Each is and must always remain dependent upon the other. In some quarters, however, there appears to be a danger of clinical methods falling into disuse, though it is unlikely that the clinical means of diagnosis will ever be wholly replaced by laboratory methods.

As a result of the movement to which I have referred, and the growth in the two directions named, treatises on medicine which attempt to deal at all fully with both the clinical and the pathological aspects of disease have come to assume very considerable dimensions. In many of them there seems to be a tendency to become more and more pathological in their arrangement and in their matter, so that students of clinical medicine and busy practitioners, whose daily work consists of an endeavour to trace from effect to cause, have been heard to complain that they do not always find in them the clinical aid they seek.

Immediately after embarking on medical practice I realised, as probably many others have done, the importance for diagnostic purposes of reviewing the various diseases which might  
ORIGIN. give rise to a patient's leading symptom or symptoms, and being unable to find precisely the information desired in any of the current text-books, I proceeded to keep a brief record of all the cases I met with, arranged under the heading of their leading symptom. This work is based upon those records, which extend

over many years, combined with the valuable knowledge imparted to me at the bedside by my teachers—more especially Dr. Charles Murchison, Dr. J. S. Bristowe, Professor J. M. Charcot, and Sir William Broadbent. Hospital clinics, at first of a general and later of a more special kind, have always been at my command; but it was at the Paddington Workhouse and Infirmary that the idea of this work was conceived, its foundations laid, and the chief part of its “skeleton” constructed. It would be hard to conceive circumstances better suited to the task, for our great poor-law infirmaries contain, as all the world now knows, a vast and almost unexplored field of every possible variety of disease, which can be studied from day to day from the beginning to the end of its course. It was, moreover, at the Paddington Infirmary that I had the satisfaction of initiating, in the year 1887, post-graduation teaching and the appointment of Clinical Assistants in workhouse infirmaries.

As regards the plan and arrangement of this work, it may be confidently affirmed that a perfect system of classification of disease is not possible in the present state of medical science, and in framing my plan, therefore, I have considered convenience and practical utility rather than an appearance of scientific precision.

PLAN.

The subject will be approached from the standpoint of symptomatology, and if I have made one principle of arrangement more prominent than another it has been that which relates to regional anatomy and the localisation of symptoms. The order of sequence will be that adopted in the examination of a patient. Thus, the first chapter will give a general scheme for the examination of a case, and will deal with certain general principles underlying methods of observation, diagnosis, prognosis, and treatment. In the second chapter the physiognomy of disease will be discussed. The succeeding chapters will deal seriatim with the symptoms and signs referable to the several organs or anatomical regions of the body, taken in the order in which they are ordinarily examined, the causes of those symptoms, and the diagnosis, prognosis, and treatment of the diseases to which they refer. Anatomy and pathology will only be referred to in their strictly practical bearings.

Each chapter will be divided into three unequal parts. Part A. will treat of the *symptoms* which may indicate disease of the organ or region under discussion, the fallacies incidental to their detection, and an account of the various causes which may give rise to those symptoms. Part B. will treat of the *physical signs* of disease in that region, and the various methods used to elicit them. Part C., which constitutes the major portion of each chapter, will be prefaced with a clinical classification of the various maladies affecting that region, and a summary of the routine procedure to be adopted; and this will be followed by a series of sections dealing with the several *diseases*, arranged according to their clinical relationships. For example, in Chapter III., on The Heart—Part A. describes and differentiates the various causes of breathlessness, palpitation, precordial pain, and the other symptoms which may be indicative of heart disease; Part B. describes percussion, auscultation, and the other methods of examining the heart; and Part C. deals seriatim first with acute, and secondly with chronic cardiac disorders, classified and arranged on a clinical basis.

Apart from the general plan and arrangement, there are two features special to this work. The first part of each chapter, dealing with symptoms and their causes, forms a SPECIAL  
FEATURES. feature on which great labour has been expended. To make each list of causes complete without redundancy, and to check the various data again and again in the light of experience, has involved an expenditure of time quite out of proportion to the space occupied. These lists will, I trust, be as useful to others as they have been to me in obtaining a clue to diagnosis.

Another feature consists of the italicised paragraphs in Part C. which stand at the head of each section which deals with a separate malady. These emphasise the salient features by which a disease may be recognised and differentiated from others belonging to the same clinical group. They are in fact brief clinical definitions, and form, metaphorically speaking, "sign-posts" or guides in the process of diagnosis. If, after carefully studying the lists of symptoms and their causes in Part A., and examining



his patient (Part B.), the reader turns to these italicised paragraphs in Part C., the work will, it is hoped, serve as a "clinical index of diseases"; for by following the plan laid down he will shortly find himself reading a description of the diagnosis, prognosis, and treatment of the malady from which his patient is probably suffering; while adjacent to this are the disorders which clinically, and very often pathologically, resemble it, and for which in practice it is apt to be mistaken.

Such an arrangement as that proposed must inevitably lead to some repetition, but this difficulty has been obviated to a certain extent by cross references. I would also ask the reader to remember that nothing fixes things so well in our minds, or aids us so much in tracing those analogies to which I shall shortly refer, as constantly looking at the same facts from a different point of view.

An attempt has been made to present the various diseases in some kind of perspective by means of different sized types, as well as by placing them as far as possible in order of importance. The relative importance of different subjects in medicine is largely a matter of opinion, and I cannot expect to escape criticism in this respect.

It is a standing accusation against medical writers that they are careless in respect to literary style, and I fear that I shall not be found an exception. I have striven to be intelligible rather than academic; and in general I fear that I must plead guilty to having endeavoured to follow the Duchess's advice to Alice in Wonderland, to "take care of the sense and the sounds will take care of themselves." When so large an area has to be covered, a certain amount of abbreviation is indispensable, and in order to condense my material it has been my practice to adopt a numerical method of description. Some may take exception to this, though the student will find it to his advantage in the acquisition of knowledge.

I may perhaps be pardoned for adverting to certain advantages which appear to me to be associated with the method that I have adopted of approaching clinical medicine. And

ADVANTAGES. first let me remark that this method of diagnosis is not what has been called a "process of exclusion." It is a

positive rather than a negative process, for by carefully considering the various causal diseases which may be in operation, and balancing the evidence for and against each, the physician is guided, not to the least improbable, but to the most probable diagnosis.

The advantages of passing in rapid review all the possible diseases which may give rise to a patient's leading symptom, are very obvious to those actively engaged in clinical work. It was Dr. Charles Murchison's method in his bedside teaching; and another equally great clinician, Dr. Matthews Duncan, has aptly remarked, "If you do not know of a thing, you are quite sure not to suspect it; and in all cases of difficult diagnosis, if you do not suspect the disease, you are almost certain not to find it."<sup>1</sup> But I am not aware that any work has yet been published which adopts precisely this plan of approaching clinical medicine.

This plan gives, I venture to think, a truer view of nature's facts than one which deals with diseases as so many separate entities. We see a case in all its clinical and practical bearings. We not only learn that the diagnosis of a patient's malady can at best be only a question of the greatest probability, but with almost mathematical precision we can also assess the probability or improbability of each of the other possible causes in operation. We learn further that all diagnoses can only be provisional, and that the degree of probability of each possible cause changes from day to day, like the coloured pattern of the kalcidoscope, as the course of the malady unfolds itself before us.

It is, moreover, in clinical work carried out on these lines—where diseases presenting analogous clinical phenomena are constantly being associated together from different points of view—that the rôle of the imagination, both in the investigation and in the treatment of disease, finds a legitimate place. The recognition of a clinical likeness between diseases has often led to the erection of a "working hypothesis" which by subsequent research has been found to be correct. Many of our greatest discoveries have been initiated in this way. It was, for instance, a process of this kind which led to the discovery that a large number of,

<sup>1</sup> "Clinical Lectures on the Diseases of Women," 4th edition, p. 15.

perhaps all, pyrexial disorders are of microbic origin. There are still a number, notably measles, small-pox, and scarlatina, in which such a working hypothesis, based on clinical resemblances, forms at present the full extent of our knowledge; but so precise are these foundations that the microbic nature of these diseases is never doubted. Hypotheses framed in this way should always be tested and confirmed in the laboratory and dead-house, whenever the morbid conditions can be produced experimentally, or when they are attended by fatal results. But unfortunately there are still a great many diseases, such, for instance, as the two great groups of clinical conditions we call hysteria and neurasthenia (conditions which form a not inconsiderable portion of the practitioner's daily work), which cannot, excepting in the most isolated instances, be observed in the dead-house, and which have not yet been produced in animals. In these cases the method of analogy or comparison to which I have just referred is not only a valuable means of investigation, it forms almost the only means we have.

It is given only to few to devote the necessary time to laboratory research; but all can study their cases at the bedside in the way indicated, and many a valuable and often unrecorded idea as to treatment will occur to the practitioner who thinks out and traces clinical analogies between diseases.

There is yet another advantage which has always appeared to me to accrue, especially to the young observer, by this process of balancing evidence and comparing diseases. It not only impresses important facts upon his memory, but it constitutes one of the best possible means of training him to habits of accurate and complete observation, and of systematic and productive thought. The scope of his horizon is widened, his faculty of systematising his knowledge becomes by practice wonderfully increased, and his reasoning powers strengthened and corrected. He finds intuitively that without accuracy in respect to the most minute details he may be led astray in the more important ones, that without system in the arrangement of his facts he will never be able to attach the proper significance and importance to each, and finally, that without judgment in attaching due weight to each item of evidence, his conclusions may be erroneous although his premises and facts are correct.

I have now described the scheme of this work, its purposes and scope—in a word, the ideal which I hoped to compass; and I believe no one could approach a task of this kind without realising the responsibilities and difficulties involved in its execution. RESPONSIBILITIES. Amidst the bewildering records of medicine there are many excellent treatises both on systematic medicine, the medicine taught in the schools, and on one or other of the several departments of clinical medicine. These deal with their respective subjects in a manner which I cannot hope to rival, and they have been to me an abundant source of instruction, but they have afforded me no exact precedent or guide along the path I wished to travel. The contemplation of the wide range of knowledge and experience required, of the immense advances which have been made of late years both in the theory and practice of medicine, of the supreme importance of the subjects here dealt with, involving as they do questions of life and death, has filled my mind with a painful sense of the obligation imposed upon me to sift my facts, and to cull my knowledge, truly, from all sources, and, before all, to obtain my material as far as possible by careful observation and patient thought from the book of nature which lay open before me from day to day at the bedside in infirmary, hospital, and private practice.

In these circumstances I have gladly availed myself of the help and advice of many friends, and there are some to whom special acknowledgment is due. In certain parts of the chapter on fevers, notably on scarlet fever, measles, diphtheria, and enteric fever, I have had much valuable advice and suggestion in the revision of the proofs from my old friend Dr. Foord Caiger. Similarly in the subject of aneurysm and in parts of the subject of pulmonary disease I am indebted to Dr. Robert Maguire, in parts of the chapter on diseases of the throat and nose to Dr. Scanes Spicer and Dr. St. Clair Thomson, in parts of the section dealing with serum-therapeutics to Dr. George Dean, in parts of the chapter on diseases of the heart to Dr. Alexander Morison, and in parts of the chapter on the urine to Dr. C. O. Hawthorne. The illustrations, with few exceptions, are taken from actual cases, and have been drawn, by the liberality of the



publishers, specially for this book under my own supervision; my grateful thanks are due to the artist, Mrs. Stanley Berkeley, a Royal Academy medallist who has lent her talent to enrich these pages with drawings which are not only accurate but, as far as scientific drawings can be, artistic. Finally, it is difficult for me to express in measured terms my indebtedness to my wife, who has assisted me in the elaboration of this work during the greater part of four years. Her skill and knowledge have largely helped to give it such completeness as it may possess; her patient industry has not only afforded me assistance, but example; and her companionship and encouragement have made many rough places smooth, and have often transformed what at times seemed to be a laborious and interminable task into a pastime.

T. D. S.

*January, 1903.*

## CORRIGENDA.

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On p. 8, Chapter XV. *should read XVIII.*, and Chapter XIV. *should read XI.*

On p. 9, Chapter XI. *should read XII.*, and Chapter XIII. *should read XIV.*

On pp. 9 and 25, Chapter XVI. *should read XIX.*

On pp. 9 and 44, Chapter XII. *should read XIII.*

On p. 18, Chapter XVII. *should read XIV.*, and Chapters XVIII. to XX. *should read XV. to XIX.*

On pp. 23, 31, 89, 93, 96, 145, 149, and 204, Chapter XIX. *should read XVI.*

On p. 36, Chapter XV. *should read XVII.*

On p. 43, Chapter XIII. *should read XII.*

On pp. 52, 76, 89, 153, 173, 179, 185, 202, and 209, Chapter XVIII. *should read XV.*

On p. 92, Chapter XVIII. *should read XVI.*

On p. 154, Chapter IX. *should read X.*

On p. 473, 3rd line from top, § 22 *should read* § 21.

On p. 473, 20th line from top, § 262 *should read* § 302.

# A CLINICAL SYSTEM OF MEDICINE.

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## CHAPTER I.

### CLINICAL METHODS.

*Preliminary Definitions — Case-taking — Methods of Diagnosis, Prognosis, and Treatment—Rules for Clinical Investigation.*

§ 1. **Definitions.** Disease is a departure from health, and it is manifested in an individual during life by symptoms. These are of two kinds—"subjective symptoms," which are recognisable only by the patient, and present no external indication, such as pain, itching, a feeling of chilliness, etc.; and "objective symptoms,"<sup>1</sup> which can be detected by the observer, e.g., abdominal enlargement, dulness on percussion, etc. The word "symptom" is used in two senses. Sometimes it is used in a general sense to indicate all the subjective and objective evidences of a disease; but more usually it is employed in a narrower sense, as synonymous with the subjective manifestations of a disorder. Confusion is obviated by using the term "subjective symptoms" when the latter sense is specially intended. Objective symptoms are usually spoken of as *signs*; and those objective symptoms which are made out by physical examination are known as *physical signs*.

Just as the value and significance of physical signs depend on the skill and experience of the physician who observes them, so the significance of subjective symptoms has to be weighed and considered in relation to the character and constitution of the patient who complains of them. Thus a certain symptom may appear trivial and unimportant to a man of strong character not

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<sup>1</sup> These words "subjective" and "objective" are borrowed from philosophy. Subjective reality is reality which exists in the mind only; whereas objective reality is that which can be demonstrated by means of tangible, visible, or outward signs.

addicted to introspection, although serious disease may be present ; whereas in a delicate woman with a susceptible nervous system every subjective symptom, however slight, may be to her a cause of great anxiety or exaggeration, and even real suffering. Submammary pain, for instance, in the first might indicate aneurysm ; in the second, hysteria.

*General (or constitutional) symptoms* are those which relate to the whole body, such as debility, or pyrexia. By some writers the term is used solely to indicate pyrexia and its concomitant symptoms, such as headache, etc.

A *latent disease* is one which is unattended by any very obvious symptoms. Thus, we speak of latent pulmonary tuberculosis when a patient suffering from tuberculosis of the lung has none of the more usual and constant symptoms of that disorder, such as cough, dyspnoea, hæmoptysis, dulness on percussion, etc. Physical signs are not necessarily absent in latent disease, but they are often difficult to detect. Some writers speak of a malady as being latent when the pain, which is usually a prominent feature of the disease, is absent. Thus, pericarditis is ordinarily attended by a good deal of pain, but this is absent in the latent form of pericarditis which frequently complicates rheumatic fever, and so also in the latent peritonitis which complicates enteric fever.

A *paroxysmal disorder* is one which comes on in the form of attacks separated by intervals of comparative health. Each attack or paroxysm consists of a stage of invasion (usually more or less sudden) leading to an acme or climax, and followed by a gradual fall or defervescence. As instances of paroxysmal disorders may be mentioned Paroxysmal Tachycardia, Angina Pectoris, Epilepsy, Nervous Faints and Flush Storms, and Paroxysmal hæmoglobinuria.

The clinical features which all paroxysmal disorders present, and a close study of those features which admit of a pathological explanation, point to the sympathetic system, and especially the vasomotor portion of it, as being probably the seat of the lesion in such cases.

The student will often meet with the termination -ITIS, and it is well to remember that it signifies "inflammation of."

**§ 2. Case-taking.** In clinical investigation, or case-taking, our object is, *first*, to elicit all the facts of the case ; and *secondly*, by reasoning based on those facts to arrive at the Diagnosis, Prognosis, and Treatment of the case. It will be found in actual practice that everything turns on the diagnosis ; that is our first and principal object ; the prognosis and treatment follow from this.



The investigation of a case consists of two parts: (A) the Interrogation of the Patient, and, (B) the Physical Examination. Students should always accustom themselves to learn all that is possible by interrogation before proceeding to the physical examination.

**A. By Interrogation of the Patient** we learn—

- a.* What is his *chief* or dominant symptom;
- b.* The facts concerning the *present illness*;
- c.* The patient's *previous history*; and
- d.* His *family history*.

Throughout the interrogation of the patient it is well to follow  
THREE GENERAL RULES:—

(1) *Avoid putting what barristers call "leading questions," i.e., questions which suggest their own answer: e.g., "Have you had a pain in the back?" suggests an obvious answer to the patient. It might be put thus—"Have you had any pain, and if so, where?"* The patient should be encouraged to tell his own story, without interruption. Moreover, the very words he uses should be recorded in inverted commas, and on no account should the words of the patient be translated into scientific terms. Some say that leading questions are permissible when the patient is very ignorant and stupid, but these are the very cases in which leading questions should be specially avoided. The only legitimate way of putting a leading question is in an alternate form, *e.g., "Have you suffered from diarrhœa or constipation?"* Time, patience, and tact are necessary to elicit the true facts of the case without irrelevant detail. Our object is to learn what the patient *feels* and knows, not what he *thinks* of his disease; and our patience is often sorely tried by a long story of his own, or his previous doctors' views on his case. Our record should be comprehensive—that is, it should include all important data, negative as well as positive—yet concise, *i.e.,* excluding irrelevant facts. Only experience and a knowledge of medicine can teach what is or is not necessary. The beginner, however, should strive after completeness rather than conciseness.

(2) A *chronological order* should always be adopted, both in eliciting and in recording the facts. Nothing is more wearisome than to wade through a mass of verbiage which mixes up dates. Dates should be recorded always in the same terms. It is very

common, for instance, to read, in students' reports, that "breathlessness began in the year 1892," "palpitation started when the patient was aged 40," "the dropsy came on three years ago."

(3) Always adopt a kindly and *sympathetic manner*. Not only is it our bounden duty to be considerate and patient with those who suffer, but by entering into the spirit of the patient's sufferings we can often get at more important facts, and a truer narration of them, than can one whose harsh or abrupt manner causes the patient to shrink up into his shell like an oyster. Put your questions in as simple and non-technical a form as possible, and be sure that the patient attaches the same meaning to the words as you do. Much will depend on the tact of the physician, and two very good rules may here be added, viz.:—Never put questions bearing on venereal disease before the husband or wife of the patient; never inquire concerning a family history of consumption or cancer before a patient whose illness is likely to be of that nature.

(a) THE CHIEF OR CARDINAL SYMPTOM.—The first question to ask a patient should always be the same: "What do you complain of?" Special attention should be paid to the symptom for which the patient seeks advice or is admitted to hospital; because it is this symptom which guides most of our subsequent inquiries. It should always as far as possible be recorded in the patient's own words. Another point in this connection is to avoid fallacies. In the following chapters I shall, after each cardinal symptom, allude to the principal conditions for which it may be mistaken. But the best way to avoid error in this respect is to verify your observations by repeating your examination again and again.

(b) HISTORY OF THE PRESENT ILLNESS.—It is better to investigate the present illness first, before the previous and family histories, because it is closely connected with the general condition and aspect of the patient, and because it generally contains more important facts. A good question to start with is, "When were you last quite well?" *Remember that the present illness dates from this to the time when the patient came under observation.* Our questions should be directed to ascertaining three facts of importance, viz.:—The precise manner of commencement of the disease, whether sudden or gradual; the date when the patient ceased to work; and the date when he took to bed. Then the

evolution of symptoms can be traced step by step in the order of their development—always in chronological order. It is useful also to know whether he has recently been, or is now, under medical treatment, not only because the symptoms may have been modified in this way, but also because one of the most important ethical principles of the medical profession may be involved.<sup>1</sup> In all of these inquiries the three general rules above given apply.

(c) THE PREVIOUS HISTORY of the patient bears largely on the etiology or *causation* of his illness, and comprises two orders of facts, viz., (1) those relating to any *illnesses* the patient may have had before the present one; (2) those concerning his personal *habits and surroundings*.

(1) Under the former heading, note in chronological order all ailments the patient has suffered from prior to the present one, with the dates of their occurrence and their duration, *e.g.*, contagious diseases of childhood; and especially such ailments as venereal disease, rheumatism, gout, etc. If the attacks have been at all obscure, it is desirable to add a few of the leading symptoms to prove the nature of the alleged attacks, and inverted commas should in such instances be freely used. For instance, "rheumatism" is a vague term which may mean any disease attended by pains in the limbs, such as alcoholism, syphilis, tabes dorsalis, or neurasthenia. The subject of syphilis should always be approached with delicacy in the case of women. Indirect information may often be gained by inquiring for prolonged sore throat, followed by loss of hair, by eruptions, or pains or swellings of the cranial or other bones. In married women a *series* of miscarriages or still-births, or children born with eruptions or snuffles, may have the same significance.

(2) The occupation, home surroundings, and previous habits as regards exercise and food should be inquired into. The daily amount of alcohol taken, and its kind (wine, beer, or spirits), should always be noted; and also the *time of day at which it was taken*, because far more harm may be done by "nips" between meals (without ever getting actually drunk) than by ten times the

<sup>1</sup> Bye-law CLXXV. of the Royal College of Physicians of London runs as follows:—"No Fellow, Member, or Licentiate of the College shall officiously, or under colour of a benevolent purpose, offer medical aid to, or prescribe for, any patient whom he knows to be under the care of another legally qualified Medical Practitioner." This is perhaps the most important guiding principle in the ethics and etiquette of the medical profession. On the other hand, this law gives us no proprietary right in a patient because we have once prescribed for him or his family. He ceases to be our patient directly the treatment stops.

quantity taken with meals. Finally, we should ascertain whether the patient has resided abroad, especially in malarial districts. In females, the previous state of the catamenia, and the number of pregnancies, miscarriages, or still-births, should be noted.

(d) THE FAMILY HISTORY may, like the previous history, have a causal relationship to the patient's malady. The age and state of health if living, age and cause of death if dead, of near blood relations should always be noted, *i.e.*, father and mother, brothers and sisters, sons and daughters. Inquiry should also be made as to whether any members of the family (parents, grandparents, brothers, sisters, uncles, aunts, or cousins) have suffered from consumption, cancer, acute rheumatism, gout, nerve diseases, insanity, asthma, heart disease, apoplexy, and especially those diseases to which the patient himself seems liable.

**B. The Physical examination** (*i.e.*, The State on admission, or The Present condition) may with advantage be prefaced by a few general remarks on how and what to observe.

(1) Here again, having learned by interrogation our patient's chief complaint, we should ask ourselves, IS THERE ANY STRIKING OR PREDOMINANT SIGN OR APPEARANCE (Latin *facies*)? The importance of INSPECTING our patient cannot be over-estimated. In these days of scientific instruments we are too apt to forget the use of our faculties. By simply using our eyes many important data may be learned besides the colour of the skin, the general nutrition, the attitude or decubitus, and the facial expression. For instance, the manner in which a patient answers questions is often the first clue to hysteria, and a peculiar mode of speech is one of the pathognomonic symptoms of general paralysis of the insane, disseminated sclerosis, and other diseases. Moreover, with experience we can by this means form a conclusion as to the kind of patient we have to deal with. Again, never be in a hurry; it is only by taking time that we can fully appreciate all the points presented to our view. This habit of "observing" the patient is only developed by long practice; but it will never be developed if the young physician allows himself to be infected by the hurry of modern times.

(2) It is important always to commence our examination with that ORGAN TO WHICH THE SYMPTOMS ARE MAINLY REFERABLE.



Some teachers direct their pupils to examine and report on the physiological systems always in the same order (first the heart, then the lungs, then the digestive system, and so forth), whatever may be the malady. But such a course has, to my mind, three objections : (i.) the student goes about the work in a mechanical fashion ; (ii.) if the patient suffer from some serious disorder, such as peritonitis, he may be seriously injured by a thorough investigation of the chest and other parts ; and (iii.) in many cases it is a waste of time to examine all the organs with equal thoroughness. The same educational advantages and experience can be obtained by the other method, and in that way we come to the most important facts first. As a general rule, the most important data should be mentioned first.

(3) In all cases EVERY ORGAN IN THE BODY SHOULD BE CAREFULLY EXAMINED ; for although we may find in one physiological system sufficient mischief to account for the patient's symptoms, the other organs may reveal changes which considerably modify our treatment, our prognosis, and even our diagnosis. Whatever order is adopted, the student should not wander from organ to organ, but examine each physiological system thoroughly before proceeding to the next. It is well to get into the habit of adopting some such order of physical examination as the following : *first*, note the general condition ; *secondly*, examine the organ chiefly affected ; *thirdly*, the other organs in the following order—Thorax (heart and lungs), Abdomen (alimentary canal, liver, spleen, and genito-urinary system), Head and Limbs (nervous and locomotor apparatus). Further details are given in the scheme below, but for the thorough investigation of the organ chiefly affected reference must be made to the chapter dealing with the diseases of that organ.

The examination should always be carried out *gently*, and *without undue exposure*. In serious cases, especially when the heart or lungs are involved, it may be well to postpone a thorough examination of some organs so as not to risk harming the patient by making him sit up. At the same time, the young physician should never allow modesty to prevent his making a thorough examination. This rule is especially necessary in patients of the better class, but a little firmness, tact, and a courteous demeanour will generally enable him to perform what is a duty both to himself and his patient.

## Scheme of Case-Taking.

### A. INTERROGATION OF PATIENT.

- (a) The patient's chief or **Cardinal Symptom**.
- (b) Facts concerning the **Present Illness**.
- (c) The patient's **Previous History**.
- (d) The **Family History**.

### B. PHYSICAL EXAMINATION. (*i.e.*, *Present Condition—* *Give date.*)

This scheme gives only the *chief points* which should be noted about the different physiological systems, with the object of excluding disease. For an exhaustive examination, such as must be made of the organ to which the patient's symptoms are mainly referable, the student should refer to the chapter dealing with the diseases of that organ.

- (a) **The general condition** may be summarised mainly under three headings: (i.) The Physiognomy or expression (especially in acute disease); (ii.) The Decubitus, Attitude, or Gait (especially in chronic disorders); (iii.) The Nutrition, General Conformation, and any Eruption on the Skin (Chap. XV.). While these data are being observed the temperature should be taken. The presence of any bedsores should always be noted.

#### (b) **Chest.**

##### I. **CARDIO-VASCULAR SYSTEM** (Chaps. III.—V.)

*Symptoms*:—Breathlessness, palpitation, cardiac pain.

*Physical signs*:—Pulse: rate, rhythm, tension, arterial wall. Heart: apex beat, percussion area, auscultation, dropsy.

##### II. **RESPIRATORY SYSTEM** (Chaps. VI. & VII.)

*Symptoms*:—Cough, expectoration, dyspnoea, pain in chest.

*Physical signs*:—Rate of respiration, inspection, percussion, auscultation, palpation.

Examine throat and nose.

#### (c) **Abdomen.**

##### III. **ALIMENTARY CANAL** (Chaps. VIII., IX., X., & XIV.)

*Symptoms*:—Appetite, discomfort after food, nausea, pain, state of the bowels, colour of motions.

*Physical signs*:—Examine mouth and tongue. Physical condition of abdomen as regards distension, and presence of fluid or tumour (inspection, palpation, and percussion).

IV. LIVER (Chap. XI.)

*Symptoms*:—Pain, jaundice.

*Physical signs*:—Size (palpation and percussion), surface (if accessible), tenderness.

V. SPLEEN (Chap. XI.)

Any enlargement (palpation and percussion) or local pain.

VI. URINARY SYSTEM (Chap. XII.)

*Symptoms*:—Any frequency of micturition, any deposit on standing, any dropsy.

*Physical signs*:—(i.) *Urine*: quantity, colour, reaction, specific gravity, albumen, sugar, deposit (microscopical examination).

(ii.) *Kidney*: Any enlargement, mobility, or tenderness.

VII. GENERATIVE SYSTEM (Chap. XIII.)

Menstruation, frequency, duration, quantity, intermenstrual discharge.

(d) **Head and limbs.**

VIII. NERVOUS SYSTEM (Chap. XVI.)

*Symptoms*:—Intelligence, sleep, neuralgia, etc.

*Physical signs*:—*Muscles*: paralysis, spasm, tremor, character of walk. *Reflexes*, deep and superficial.

*Sensation* for touch, pain, temperature.

*Cranial Nerves*:—Vision, pupils, movements of eyes, fundi. Movements of face, tongue and palate.

Hearing. Smell. Taste.

*Sympathetic System*:—Flush storms, trophic lesions, obscure sensations.

(e) **Blood.** In anæmic and some other cases the blood must be examined (Chap. XX.).

**Progress of case.** Notes (daily, of acute or febrile cases, twice a week of sub-acute, and once a week of chronic cases), should be made of the progress of the case; and much care is required here to avoid redundancy on the one hand, and on the other to record completely all important changes, or any fresh symptoms, and the effect of the treatment adopted. In acute febrile cases there ought to be a daily note, and the pulse, respiration and temperature should be noted several times daily. In chronic cases it will be sufficient to note, once a week, the persistence of the prominent symptoms or any change in the symptoms. In all cases any sudden change in the patient's symptoms or general condition should be at once noticed. Each note should have special reference to the previous one; and before taking a fresh note, the previous one should be read over. The treatment and its effects should always be incorporated; thus, if the patient has been ordered diaphoretics, purgatives, etc., record should be made of the state of the skin, bowels, etc.

**History sheets, charts, diagrams, etc.** A history sheet for recording the history of a patient should be ruled with one vertical line down the page one-third from the left-hand margin, so as to give space for information learned subsequently. It should have printed headings and spaces at the top, thus:—

**Diagnosis.** (Space here for primary and secondary disease, filled in by physician afterwards.)

**Name**..... **Age**..... **Sex**..... **Occupation**.....

**Address**..... **Date of admission**.....

**Chief symptom on admission**.....

**Date of discharge**.....

Temperature charts are of the greatest use to record the temperature and other facts of diurnal variation.

Outline diagrams of the various regions of the body are now prepared, and are very useful.

A kind of shorthand code for physical signs is advocated by some authors, and, when once learned, may be useful in saving time and space.



§ 3. **Examination of children and infants.** Here the same general rules apply as to interrogation and physical examination, and we should first endeavour to ascertain the child's leading symptom, either from the patient or its relatives. There are, however, certain additional rules upon the adoption of which much of our success with children will depend.

1. First endeavour to establish friendly relations with your little patient. This may be done sometimes by appearing not to notice the child when you first enter the room; after a while it may make advances and investigate your watch chain or ring, etc. Time should always be given for the child to become accustomed to your presence, and anything like abruptness will defeat your aim entirely.

2. The questions put to the child should always be of the simplest character—e.g., "Where does it hurt you?" From the mother you may learn the age up to which the child remained healthy, the symptoms of the present and previous illnesses. In the case of an infant ask whether it was a full-time child, if born with instrumental aid, whether it was born healthy, or whether it developed a rash or "snuffles," and whether breast or bottle-fed. If the child is past early infancy the same questions may still be put, and in addition inquire when it began to walk, and when dentition commenced. Carefully inquire as to its present and past diet, as to its appetite, and the state of the bowels. Ask also how long it sleeps, bearing in mind that children require much more sleep than adults. Then inquire for any recent illness in other members of the family.

**PHYSICAL EXAMINATION.** Valuable as *attentive observation* may be with adults, it becomes quite indispensable with children, who cannot accurately describe their sensations. Much may be learned while you sit and allow the child to get accustomed to your presence. Notice its expression, the brightness of its eyes, its attitude, the colour of its skin, the state of nutrition, its size as compared with age, its movements, the condition of its lips (moist or dry), the character of the breathing, the sound of its voice. If it cries, inquiry should be made whether this is constant or only at times. Congenital syphilis may be plainly depicted on its face or skin. If the child be asleep when first you enter, do not wake it, but notice all the above before it is disturbed. The limbs of a healthy child should be constantly on the move; drowsiness, dulness and listlessness are signs of pyrexia, and especially of the contagious fevers. The hands are instinctively moved towards a seat of pain—e.g., the head in meningitis. The state of the temper is altered in the prodromal stage of most diseases; but it is markedly peevish in the prodromal stage of meningitis. For other facial alterations see **Facies** (§ 12). When the child is undressed for examination the back of the chest should be examined first, while the child looks over the mother's shoulder at some one who attracts its attention with a bright object or a bunch of keys. The binaural stethoscope is the most useful under these circumstances.

§ 4. **Methods of Diagnosis, Prognosis, and Treatment.** Diagnosis, prognosis, and treatment are the objects we had in view in eliciting all the facts concerning the patient by the process of "Case-taking." Of these three, **Diagnosis**—which as the Greek word (*διαγνωσις*) implies, means the distinguishing

or discernment of the disease—is by far the most important. Everything necessarily hinges on that, because without the recognition of the disease, rational prognosis and treatment are impossible. It will be well, therefore, to consider how the facts we have elicited may be utilised in order to arrive at a diagnosis. Several different methods are employed:—

The method usually adopted, which is the outcome of the student's studies in systematic medicine, is to erect a *hypothetical diagnosis*, and to see whether the patient's symptoms tally with the description of the disease. When a child, for instance, with disorderly movements comes before us, the diagnosis of chorea at once occurs to our minds. The age of the patient, character of the movements, and all the obvious features of the case appear to correspond with that disorder. It does not seem necessary to consider any other suggestion. This method answers well enough in straightforward, well-marked, typical cases; but in cases presenting anything unusual or atypical considerable difficulty may be experienced.

Another method of making a diagnosis is by a *process of exclusion*; that is, after studying the diseases which might possibly be in operation, we arrive at our diagnosis by excluding those which the disease least resembles. In such diseases as enteric fever, where the symptoms are few in number, this may be the only method possible. The patient, for instance, is suffering from a moderate degree of pyrexia, the illness came on gradually; that is all we may know about the case. There are many possible causes of such a condition, but we arrive at the conclusion that it is probably enteric fever, because all the other possible diseases are rendered improbable for one reason or another.

The third method consists of *balancing the evidence* for and against all the possible causes (clinical or pathological) which might give rise to the patient's leading or cardinal symptom (such as headache, albuminuria, or vomiting). In this method, after having elicited all the facts of the case, we return to the patient's *cardinal symptom*, enumerate in our own minds the various causes which might give rise to that symptom, and balance the evidence adduced by the other facts of the case for and against each one in turn. It may strike some as being a little tedious,

but it is not so when we have got into the habit of employing it. It is certainly the one best adapted for the elucidation of obscure or atypical cases; and under all circumstances it presents a truer picture to our mind, because diagnosis can never be a matter of absolute certainty. At most a diagnosis is only a strong probability, and this method enables us to ascertain the exact amount of probability in each disease. Even in the simplest and most typical cases it is a good mental exercise for us to keep in mind the other lesions which might produce the same symptom, and then we are always on the look-out for possible errors, and ready at any moment to review the diagnosis—a correct mental attitude when in presence of nature's phenomena. The chapters which follow are based on this last-named method.

EXAMPLE. Let us suppose, for instance, that the patient, a pale young woman, aged 23, comes to us complaining of **vomiting blood** (*i.e.*, hæmatemesis).

First, we ascertain and verify this, the leading symptom, and find that she has really vomited a considerable quantity of blood.

Secondly, we INTERROGATE her as to the history of her present illness, her previous and family histories, and we find that she has suffered for several years from symptoms pointing to dyspepsia, and that latterly there has been severe pain in the epigastrium. There are always four features we have to investigate about every pain—its position, character, degree, and constancy; and we find that this epigastric pain is a sharp pain, not constant, but coming on shortly after taking food, and that it is followed *and relieved* by vomiting. The other details of the case we will omit for the sake of brevity.

Thirdly, we proceed to the PHYSICAL EXAMINATION, first of the abdominal organs, but this reveals nothing abnormal. Then we go through the other physiological systems in order, observing (a) her General Condition (noting, for example, how pale and thin she is, and how weak she seems); (b) examining the Chest (cardio-vascular and respiratory systems); (c) the Head and Limbs (nervous system); (d) the Blood must also be examined, because anæmia (poverty of blood) may be inferred from the pallor of her skin.

Having elicited all the facts (taken the case) by interrogation and physical examination we return to the *cardinal symptom*—hæmatemesis<sup>1</sup>—and consider its various causes (see the section on Hæmatemesis) *seriatim*, taking the most probable cause in this case first.

<sup>1</sup> Here there was no difficulty about identifying or selecting which was the chief or most important symptom; but in another case the anæmia (or the vomiting or epigastric pain) might be the more serious or prominent symptom, the hæmatemesis consisting, perhaps, of a few streaks of blood. Then we should deal with the anæmia in the same way as hæmatemesis is here dealt with. Sometimes a good deal depends upon our choice of which is the "leading symptom," for it is not always the most prominent which is the most serious and important; and by an error in this respect we may be led far afield of the true disease. Sometimes, however, it is useful to change the point of view we take of a case, by regarding it from another standpoint or leading symptom.

## (a) SIMPLE ULCER OF THE STOMACH.

*For*: (i.) the profuseness of the hæmatemesis; (ii.) the character of the pain (brought on by food, relieved by vomiting); (iii.) the history of dyspepsia; (iv.) the age and sex of the patient.

*Against*: (i.) no tenderness in the epigastrium.

## (b) CANCER OF THE STOMACH.

*For*: (i.) the vomiting of blood; (ii.) pain in the stomach; (iii.) pallor and emaciation; and so on.

*Against*: (i.) the blood vomited was too profuse and had not the character special to cancer (coffee grounds); (ii.) the pain was only produced by food and entirely disappeared after vomiting; (iii.) age of patient much too young.

## (c) PORTAL OBSTRUCTION.

*For*: (i.) the profuseness of the hæmatemesis.

*Against*: (i.) absence of abnormal signs in the liver; (ii.) absence of ascites, piles, and other symptoms of portal obstruction.

(d) OTHER AND LESS PROBABLE DIAGNOSES can be discussed in like manner, though each of these may be more summarily dismissed thus: *Vicarious menstruation* would not account for the dyspepsia, acute epigastric pain, and other symptoms. *Leucocythæmia*, *Scurvy*, and *other blood conditions*, if present, would present the other symptoms of those maladies; and so on.

It follows, therefore, that the balance of evidence is in favour of (a) SIMPLE ULCER OF THE STOMACH, partly because of the weighty arguments in its favour, and partly because the only argument against it is not vital, for tenderness may be absent when the ulcer is situated on the posterior wall of the stomach. Indeed, if a numerical value were given to each of the "reasons" for and against, it would be possible to express the precise degree of probability in each disease in the form of a mathematical ratio. This method may at first sight seem tedious, but after a little practice it becomes automatic and extremely simple; and it takes much less time than is here implied.

**Prognosis** (from the Greek word *προγνωσις*) is a "foreknowledge" of the events which will happen—i.e., of the probable course the disease will run. Nothing but wide experience, combined with careful and minute observation, will enable a physician to prophesy with any approach to accuracy. It will, however, be useful to bear in mind that the prognosis of a case depends upon four circumstances, viz., (1) the *usual course*, duration, and event of the disease in operation (phthisis, for instance, runs a prolonged course, and until lately the event was almost invariably fatal); (2) the presence or absence of *untoward symptoms* (e.g., profuse hæmoptysis in phthisis); (3) the presence or absence of *complications* (which are sometimes more fatal than the disease itself—e.g., enteric and



many other fevers are fatal chiefly by their complications); and (4) the *causes* which are in operation, including among the predisposing causes such data as age and sex (bronchitis, for example, in middle life is not a serious affection, but in infancy and old age it is one of the most fatal diseases in the Registrar-General's returns). More reputations are wrecked on the rock "prognosis" than on any other.

As practical hints to the young physician I would advise him (1) never to commit himself to a prognosis unasked, or before the effects of treatment have been noted. (2) It is also well to impress upon the friends that a "physician" cannot hope to be also a "prophet"; and that prognosis may depend on many factors in the case which are not yet revealed. The medical work for Life Insurance is largely a question of prognosis.

**Treatment** is what the patient comes to us for; and it may be of three kinds: (1) In *Radical* treatment (also called Curative or Rational) our object is to cure the patient of his disease by the removal of the cause. This is the only truly scientific treatment, and it is based mainly upon a knowledge of the pathology of the malady. (2) *Symptomatic* treatment is directed only to the relief of the symptoms. In some incurable maladies, symptomatic treatment is the only kind that is possible, and all that we can do is to ease the passage to the grave. But in the practice of busy practitioners, the trouble and time needed for thorough investigation often lead to the adoption of the latter at times when a more radical treatment would be possible. There is an unfortunate tendency to fall into a routine of symptomatic treatment which we should constantly guard against. Both Radical and Symptomatic treatment may be either internal or external on the one hand, and either medicinal or dietetic and hygienic on the other. (3) *Preventive* treatment has within the last quarter of a century developed almost into a separate science, the science of Hygiene or State Medicine.

§ 5. **General Rules in Clinical Investigation.** There are certain habits which the student should strive to cultivate when he comes to the practical aspect of his profession; and he should remember Thackeray's saying: "Sow an act and you



reap a habit; sow a habit and you reap a character; sow a character and you reap a destiny." Clinical medicine depends more than anything else on accurate, complete, and well-directed observation, and there are five hints I would give to the student in this connection.

1. *Avoid superficiality* in your observations. Do not try to see many cases in one day, but rather one or two cases *continuously from day to day*, so that you may follow a given malady throughout its entire course. It is of more value to follow up one case in this way than to see a dozen on one occasion only. Practical knowledge must be acquired gradually. The thought will often occur to the student how slowly he progresses with his clinical knowledge. This is partly real, partly apparent. It is partly apparent because a student does not realise at the time the value he derives from listening, for example, to the same cardiac murmur over and over again. It is partly real because it is only by patiently devoting the necessary time to the study of the same case from day to day that he will learn to make his observations adequate, thorough and precise. That is why many a brilliant intellect falls behind, and many a plodder comes to the front in our profession. It is vain to attempt to substitute genius for patient industry in this arena. You must learn for yourself the effects of this or that line of treatment; learn to correct and control the observations you make one day by your observations of the morrow; and above all try to learn what is the sequel or termination of the case, especially in such instances as may lead you to the deadhouse. There, more than anywhere else, the most brilliant diagnosticians learn from their own errors more than from a multitude of successful cases.

2. *Do not strive after what is rare and curious.* It follows, as a matter of course, that, other things being equal, a fact is more important in proportion as it is common. Moreover, by studying *only* the exceptions to a rule, our minds will have a distorted view of clinical phenomena. Do not, therefore, be led astray by those pedants who seek after the singular and uncommon. It is well to see rare cases when the opportunity offers, by all means, but be careful that you mentally register them as rare.

3. *Do not study only acute and severe cases.* It is true that in

acute diseases there is often more to be done, more heroic and decisive effects to be produced, or apparently produced, and therefore more credit and renown to be obtained. But we shall find in actual practice not one-tenth, perhaps not one-hundredth of our patients will be suffering from these complaints. Our success therefore in practice, whether measured by that laudable satisfaction at having done one's duty, or by the pecuniary reward of which every earnest labourer is worthy, will depend very much on our experience of, and our ability to treat chronic, and what we are too apt to call trivial complaints. For one case of pneumonia, or Addison's disease, the student will, I venture to think, have a hundred cases of dyspepsia, chronic rheumatism, or chronic bronchitis. In the treatment of such complaints the greatest judgment and thoroughness are sometimes needed. No sudden or startling effects can be produced. Chronic diseases require chronic remedies, and it is only by experience that one can learn to produce those gradual effects which lead to a successful issue.

4. *Be accurate in your observations.* State facts precisely as you find them, no matter whether they accord with your hypothesis or not; and state only what you find and know to be the truth. The study of clinical medicine, like the study of any other of nature's phenomena, should inculcate in the mind of the student a love of truth. It is impossible to have any dealings with nature without learning that truth is the key to the discovery of her secrets. Accuracy is one form of truth, and it is only by repeatedly going over your observations, and sifting the patient's statements, that you can ensure accuracy.

5. *Be systematic in the arrangement of your facts,* for it is only by a systematic arrangement that you can attach the proper significance and importance to each, and get a firm grasp of the whole case. Nothing, for instance, is more liable to confuse and to prevent you coming to a correct conclusion than wandering from one date to another without regard to the chronological sequence in the history of an illness. And again, in physical examination nothing is so likely to lead you astray as wandering from organ to organ without first completing the examination of each.

### § 6. Classification of Diseases — Method of Procedure.

It has been customary, and the practice is convenient, to classify diseases into two great groups—Constitutional and Local. LOCAL diseases are those in which the principal and perhaps the only lesion is localised in one organ or situation. CONSTITUTIONAL diseases are those which affect the blood and therefore the whole of the body; a considerable number of these are now known to be microbic in origin. As illustrative of the latter, acute rheumatism, typhoid fever, pyæmia, and anæmia, may be mentioned; of the former, iritis, synovitis, and myelitis.

It is convenient for clinical purposes to preserve this division, but the rapid advance of pathology has gradually transferred disorders from the "local" to the "constitutional" group. A large number of diseases, formerly believed to be lesions of local origin (such, for instance, as pneumonia, endocarditis, and peritonitis) are now known to be due to some general morbid process, toxic or microbic, having a special local manifestation.

From a pathological standpoint diseases are sometimes divided into 2 groups—Organic, those in which some anatomical change is found after death, and Functional, those in which no structural alteration is found. The anatomical or structural change is spoken of as the "lesion." The word Functional must not be regarded as synonymous with Hysterical.

Now it so happens that local disorders are very often met with as complications or effects of constitutional or general conditions; and since in clinical work we are engaged in **tracing from effect to cause**, we shall in the following chapters take the local diseases which are manifested by a lesion *localised* in some particular organ first, and the *constitutional* conditions afterwards.

When a patient applies to us, if, as the result of our inquiries, we find he is suffering from a symptom localised to some organ (*e.g.*, pain in the liver), turn to the chapters relating to the diseases of that organ (Chapters III. to XVII.).

If, on the other hand, he has no localised symptom, but complains of malaise, feverishness, or a sense of "bodily illness," turn to constitutional diseases (Chapters XVIII. to XX.).

## CHAPTER II.

### THE FACIES, OR EXTERNAL APPEARANCE OF DISEASE.<sup>1</sup>

IN our scheme of case-taking it will be remembered that the first step in physical examination was to observe the patient's general condition; and it will also be remembered how great was the importance to be attached to an adequate inspection of the patient while he was telling us the story of his illness.

Some diseases can be identified almost at a glance, before the patient opens his lips, such, for instance, as Chronic Alcoholism, some manifestations of Hereditary Syphilis, Graves' Disease, Cretinism, Myxœdema, Facial Paralysis, and Hydrocephalus, when these conditions have passed beyond the incipient stage. The existence of others can be very strongly suspected, such as Rickets, Post-nasal Adenoids (mouth-breathing children), and Chronic Bronchitis with Dilated Right Heart.

But, apart from these, much may be learned from the first glance at a patient—from his *decubitus* (the way in which he lies), from his *attitude* or gait (if he be able to leave his bed), from the expression of his *face*, the colour of his *skin*, and from the *general conformation* of the body—without the employment of any special methods or apparatus for diagnosis. It is to be feared that as scientific methods become more and more perfect, these means, which constitute one of the most useful and important aids to diagnosis and prognosis to the experienced busy practitioner, are apt to be neglected. But, on the other hand, students and young practitioners had better not attempt "lightning diagnoses," or they will certainly fall into the most serious errors. Some men, it is true, like the late Dr. Sibson, seem to be specially gifted in this way; but it is only by long experience

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<sup>1</sup> The Latin word *facies* signifies an appearance, form, or shape.

and the possession of special faculties that they can accomplish such feats.

It is a fundamental rule that your patient should face the light at all medical interviews. Similarly your own chair should be in the shade, lest the patient should read too readily what is passing through your mind. It is surprising what important clues can be obtained by an intelligent inspection of your patient, both as to his character and his disease. The facies of disease may be summarised under three headings: (A.) THE PHYSIOGNOMY OF DISEASE. (B.) THE DECUBITUS, ATTITUDE OR GAIT. (C.) ALTERATIONS IN THE GENERAL CONFORMATION OF THE BODY.

Hints to be derived from an inspection of the hands are given under diseases of the extremities. With the exception of Hereditary Syphilis, which produces such indelible marks on the surface of the body, the various diseases will be only mentioned here. The description and differentiation of the several affections referred to will be entered into more fully in the chapters which follow.

#### (A.) THE PHYSIOGNOMY IN DISEASE.

An observant physician can obtain important clues to diagnosis by the physiognomy, *i.e.*, the aspect and expression of the patient's face, even apart from the insight which can be gained by this means into his character.<sup>1</sup>

§ 7. In **acute diseases** more can be learned from the position in which the patient lies (*i.e.*, his Decubitus, § 14) than from the physiognomy or expression of his face. But it is worth remembering that the face assumes an *anxious expression* which is very characteristic in pericarditis, peritonitis, and severe pneumonia; also during attacks of angina pectoris. The supervention of *acute pericarditis* in the course of rheumatic fever is often unsuspected, as there may be no local symptoms; but it may be recognised by this anxious expression, the dilated nostrils, and the flush upon the cheeks which were (probably) at our last visit so pale. In *acute croupous pneumonia* again the appearance is very distinctive. The flushed

<sup>1</sup> Those who are interested in this method of studying character should consult "Essays on 'Physiognomy,'" by Lavater, translated by Thomas Holcroft (Ward, Lock & Co., London), 1783.



face, hot dry skin, widely dilating nostrils, the eruption of herpes beside the mouth, and the profound disturbance of the pulse-respiration ratio (1 : 2 instead of 1 : 4, which is the normal), form a picture which greatly aids the recognition of the disease. The *Facies Hippocratica*—a facies or appearance, of which the description has been handed down from Hippocrates—is the forerunner of death from exhaustion, such, for instance, as the final stage of cholera, and wasting disorders. The temples are hollow, the eyes sunken, the eyelids slightly parted, the eyes glazed, and the lower jaw droops. The *Risus Sardonicus* is a fixed grin, met with most typically in tetanus. The corners of the mouth, which twitch at intervals, are drawn upwards as in laughter, and the features assume a fixed sarcastic expression. In several abdominal disorders, such as peritonitis, a similar expression occurs, in which the curved folds stretching from the alæ nasi to the corners of the mouth become marked.

§ 8. A few **chronic diseases** may be enumerated in which the physiognomy is characteristic.

(i.) The aspect of a *phthisical or tuberculous* patient differs in the premonitory and advanced stages. (a) Before any evidences can be detected by physical examination of the chest, the patient has the appearance which is loosely described by the laity as “delicate.” The skin is fine and soft, and the fresh, rosy colour of the cheeks is out of keeping with the dark rings around the sunken eyes. But it is by the deficient chest measurements and sloping shoulders that the “strumous diathesis,” as this tendency or predisposition to tuberculosis is called, makes itself especially manifest. The shoulders slope, and the transverse diameter is deficient in proportion to the antero-posterior. (See Chapter VI.) Sometimes such patients are plump and rosy, nevertheless they have a deficient chest measurement. (β) When the disease is advanced, the phthisical patient often presents an appearance that enables the physician to hazard a diagnosis almost without further investigation. The pale emaciated face, with sunken eyes, the circular crimson flush of hectic fever on the cheeks, the wasted body bathed from time to time in sweat, the hoarse voice and easily provoked dyspnoea, collectively form a picture which is very characteristic.

(ii.) *Chronic Bronchitis with Dilated right heart* is another condition of extremely common occurrence in the practitioner's daily practice, and the picture these patients present is very characteristic. The florid "healthy" looking cheeks, the pulsating jugulars, in a person over forty (more often of the female sex), is very typical. There is mostly a history of winter cough for several years.

(iii.) In *chronic alcoholism* there is a puffiness of the face and a congested watery look about the eyes ("a blear-eyed look"). The eyelids are puffy, and the person is well described by sailors as having "an eye like a poached egg." The cheeks and nose are often red, and dotted with stellate venous capillaries. The belly is corpulent; and on holding out the hands and spreading the fingers, they are seen to be affected with fine small rhythmical tremors. The whole picture is unmistakable, though the eyes alone will tell the tale.

§ 9. **Swelling of the face** and neck, if associated with œdema of the limbs and trunk, may be part of the generalised *dropsy of renal disease*: but on account of the looseness of the cellular tissues around the eyelids it is most obvious in that situation. The puffiness of the eyelids due to renal disease is, however, greater in the morning than in the evening; and in this way may be distinguished from a similar condition due to arsenical poisoning or whooping-cough.

A swollen œdematous condition of the face accompanied mostly by a troublesome redness, coming on after meals, is a symptom for which *dyspeptic patients* often seek advice. It also forms part of that troublesome condition *urticaria factitiosa*. A swelling of the face is also apt to occur with different forms of erythema, and is generally worse after meals.

*Chronic œdema around the eyelids* must not be mistaken for Myxœdema. It is a not infrequent sequel to recurrent eczema, or repeated attacks of erysipelas, in that situation. It is also met with in nervous or hysterical conditions, and in vasomotor derangements.

Edema localised in the head and neck only is found in those rare cases where there is pressure on the *veins within the thorax*, especially the Superior Vena Cava, as in cases of mediastinal tumour.

*Myxœdema* may often be recognised by a glance at the patient's face and hands (Fig. 1). There is a solid œdema and puffiness of the face—the body and limbs being also affected—but it does *not pit on pressure*. The vacant, stolid look, flushed cheeks,



Fig. 1.—MYXŒDEMA. The patient was a man aged 30, who was admitted into the Paddington Infirmary in November, 1887, presenting all the usual symptoms of the disease. His movements and mental processes were extremely slow, average temperature 97·2; the quantity of urea passed was less than half the normal. There was a tendency to hemorrhages. (See further particulars "Med. Soc. Proc.," vol. xi.)

scanty hair and slow speech are equally typical of this disorder. The hands are flat, coarse, and swollen. (See Chapter XIX.)

§ 10. The **complexion** and colour of the face will repay careful inspection; and the trained observer will acquire some useful hints. Thus, the *pallor* of anemia, syphilis, or tubercle, and other anæmic conditions (Chapter XIX.) is often very striking. So also is the pallor or rather *sallowness* of aortic

valvular disease; the dead white or *waxen puffy* appearance of parenchymatous nephritis; the *greyish pallor* of chronic interstitial nephritis; the *greenish* colour of chlorosis, the *primrose* colour of that happily much rarer condition, pernicious anæmia.<sup>1</sup> The *orange* colour of jaundice; the *mahogany* colour or *bronzing* of Addison's disease; the dull *earthy* look of malarial cachexia,



Fig. 2.—EXOPHTHALMIC GOITRE (Graves' Disease).  
From Byrom Bramwell's "Atlas of Clinical Medicine."

cancer, and chronic abdominal disease; and finally, the *purple* (or cyanotic) appearance of the cheeks and lips in mitral and congenital heart disease, are still more distinctive. *Dark rings* around the eyelids appear in states of fatigue; they often indicate want of sleep, or indigestion, and may be so pronounced in malarial conditions as to resemble the ecchymosis of a bruise.

Seborrhœa Oleosa of the scalp gives rise to *greasiness of the face*; and this with the erythema which frequently accompanies it, produces an appearance somewhat resembling a

badly polished copper kettle. A greasy complexion of this kind is a great affliction to some young and otherwise attractive women, who might easily be rid of it by the cure of the scalp lesion. A *muddy* sallow complexion may be associated with dyspepsia, when the lips are usually dry; but this also may be another result of seborrhœa of the scalp of the kind just mentioned.

<sup>1</sup> It is only by long experience that one is enabled to distinguish these refinements of shade.



§ 11. The **Face in Detail** merits a little closer study ; and first, that most eloquent portion of it, the eyes.

(i.) The *Eyes* may be *protuberant* as a whole (Proptosis) in Graves' disease, intraocular tumour, etc. (Chapter XVI.). Protrusion of the eyeballs is the most constant symptom of Graves' disease. An equally common sign of this disorder is a goitrous enlargement of the thyroid gland, and therefore the malady is also called Exophthalmic Goitre (see Fig. 2). The eyeballs may recede in paralysis of the cervical sympathetic, in wasting diseases, collapse, and the diseases which lead to collapse.<sup>1</sup> In the *sclerotic* or white of the eye the tinge of *jaundice* can often be detected when the yellow colour of the skin is so slight as to escape detection. The "*arcus senilis*" is a white ring of opacity in the cornea, just within its peripheral margin. It is usually believed to indicate senile degeneration of the arteries and other tissues of the body, but I never found it so among the old people in the Paddington Workhouse and Infirmary. In adults who are the subjects of hereditary syphilis, the cornea may present *striae*, or the appearance of ground glass (Fig. 3), due to interstitial keratitis. Alterations of the *pupil* are dealt with elsewhere (Chapter XVI.).



Fig. 3.—HEREDITARY SYPHILIS. Showing the interstitial keratitis and "pegged teeth" of Hereditary Syphilis.

(ii.) The *lips* may show the pallor of anæmia on the one hand

<sup>1</sup> See also Treacher Collins, *Brit. Med. Journ.*, 1899, p. 846.



or the congestion of cardiac disease on the other. Fissures and mucous tubercles may indicate that syphilis is in operation. Stellate cicatrices around the lips are a record of previous or hereditary syphilis.

(iii.) The *teeth* also may present the evidences of hereditary syphilis, in which disease, as Hutchinson has pointed out, the permanent incisors (that is to say, when the child has reached the age of seven) are characteristically "pegged" (see Fig. 3, p. 25, and §§ 17 and 144).

(iv.) Depression of the *bridge of the nose*, if marked, is due to chronic rhinitis in childhood, usually of syphilitic origin. In such cases the nose is characteristically broad and flat, or small and "snub," like a button.

§ 12. The **Physiognomy of childhood** requires considerable experience to appreciate it fully; then it lends us invaluable aid.

(i.) *Congenital syphilis* gives to an infant a very characteristic, pinched, wan, or "senile" face. The complexion is ashy-grey, the skin is "drawn," and it may be flaky or parchment-like. The eyes and cheeks are so hollow that the nose seems unduly prominent, and thus gives to the infant the appearance of a little wizened old man. The wasted frame and other signs are mentioned below (§ 17).

(ii.) When an infant is experiencing *pain* the face will sometimes give a clue as to its situation. Thus, a wrinkling of the forehead or frown is indicative of pain in the head; a drawing up of the mouth at the corners, producing marked naso-labial folds, points to severe abdominal pain; a dilatation of the nostrils and elevation of the eyebrows may suggest intra-thoracic discomfort; and in *Tabes Mesenterica* and other chronic wasting diseases the face gradually assumes a fixed or contracted condition in which the angles of the mouth are depressed.

(iii.) The *fontanelles* afford information as to the general condition of a child. A *depressed* fontanelle is an untoward sign in all acute illnesses of childhood—*e.g.*, the diarrhœa and vomiting of infancy. The fontanelles *bulge* in congestion of the meninges, and this is a useful diagnostic feature between true meningeal affections on the one hand, and fevers, broncho-pneumonia, and other

diseases with cerebral symptoms on the other. The fontanelles are tense and bulging in all diseases causing increased intracranial pressure—e.g., cerebral tumour. Normally the fontanelles should be closed between the ages of one and a half and two years. In Rickets they are late in closing.

(iv.) Nothing is more characteristic than the *listless* and apathetic facies of children suffering from the early stages of fever.

(v.) *Rickets* presents pallor, but nothing more distinctive in the face until the later stages are reached, and the child is three or more years old. Then the prominent and rounded forehead and other signs appear.

(vi.) In *mouth-breathing children* (due generally to post-nasal adenoids), when they reach the age of about ten to fifteen years the expression is very characteristic. The broad bridge of the nose and open mouth give to them a vacant, stupid appearance, which sometimes belies their intelligence; though sometimes they are, in fact, mentally backward.

§ 13. **Variations in the form of the skull** are met with in several complaints; and chiefly in children, because cases of marked deformity of the head seldom reach adult life. It may be remarked, in the first place, that a small degree of *asymmetry* of congenital origin is of fairly common occurrence.

(i.) In *hydrocephalus* ("water on the brain") the head is enlarged generally, dome-shaped, and with wide fontanelles; and the face appears relatively small (see Chapter XVII.).

(ii.) In *hereditary syphilis* the bones around the anterior fontanelle are thickened, and irregular patches of thickening and thinning are met with. In children this thinning most frequently occurs in the parietal and occipital regions, and is known as *craniotabes*. It is generally an indication of rickets in association with infantile syphilis.

(iii.) In *rickets* the vertex is flat, the forehead prominent, and there are bosses on the frontal and parietal regions.

(iv.) The head is unduly small in *microcephalic*, and boat-shaped in *scaphocephalic* idiots. In the former the cranium is disproportionately small; in the latter the antero-posterior diameter is elongated and its lateral diameter diminished. The palate is generally high and arched in both.

(v.) Localised thickenings of the bones of the *adult skull* occur in hereditary syphilis, rickets, osteitis deformans, leontiasis ossea, and after injury.

(vi.) In *Acromegaly* (see Diseases of the Extremities) the lower jaw is enlarged, and sometimes the nose also. In this condition the face is ovoid, with the long transverse diameter below. In *Paget's disease* (osteitis deformans) it is ovoid, with the long transverse diameter above: the hands and feet are also big and clumsy, but the skin is normal.

In various diseases of the nervous system the face presents a pathognomonic and typical expression. Thus in Bell's or *Facial paralysis* the face is distorted, and so also in that rare condition *facial hemiatrophy*. The expression is *vacant* in idiocy, some hysterical subjects, and early disseminated sclerosis. A smooth *expressionless* appearance (differing from the preceding in that there is a lack of mobility) is very characteristic of paralysis agitans, and, among rarer conditions, double facial paralysis, sclerodermia, and Raynaud's disease (on account of the sclerodermia present). Bulbar paralysis gives a very characteristic, mournful or sullen appearance to the face. In this disease the orbicularis oris is paralysed, and allows the lower lip to pout; while the weakness of the zygomatici results in a drooping of the corners of the mouth such as we usually associate with sorrow, or sullenness of temper. In a more advanced stage the saliva dribbles out of the mouth.

(B.) DECUBITUS (IN ACUTE CONDITIONS) AND ATTITUDE  
(IN CHRONIC DISEASES).<sup>1</sup>

§ 14. **Decubitus** signifies the position in which a patient tends most constantly to lie, and it often gives a valuable clue to the disease, more especially in the diagnosis of **Acute Diseases**, and sometimes as to their probable issue as well. For example:

(i.) *Sitting up in bed*, propped up with pillows, on account of inability to breathe in another position (orthopnoea), is characteristic of the extreme breathlessness which occurs in advanced cardiac, pulmonary, or renal disease.

(ii.) *Lying on one side* is characteristic of considerable pleural effusion or pneumonia on that side. When a phthisical patient always lies on one side, we may suspect a cavity, bronchiectasis, or empyema of that side.

(iii.) The *dorsal decubitus*, i.e., lying on the back, is seen in grave illnesses attended by marked prostration. In the "typhoid state" the limbs are stretched out and completely relaxed. But if the prostration be due to peritonitis the legs are drawn up so as to relax the abdominal muscles; and for the same reason the breathing is thoracic, and the abdomen is quite still. The typhoid state, so called from its frequent occurrence in typhus and typhoid fevers, is a condition of profound prostration, attended by unconsciousness or muttering delirium, sordes on the teeth, and dry cracked tongue.

(iv.) *Opisthotonos* is an arching of the back which occurs in some

The various characteristic gaits are described under Diseases of the Nervous System.

convulsive and spasmodic disorders. It may be so great that only the head and heels touch the bed. It is met with in tetanus, hystero-epilepsy, and cerebro-spinal meningitis.

(v.) *Restlessness* occurs in many disorders, acute and chronic, and is generally a grave sign in the former—*e.g.*, in acute pericarditis. Sometimes, as in children, it is an indication of severe pain. *Carphology* (καρφος = the clothes, λέγειν = to pluck), or floccitatio, is the picking at the bedclothes so characteristic of the “typhoid state.” The hands seek after imaginary objects. *Subsultus tendinum* is the muscular twitching or tremor which occurs in the same state. Both of these imply extreme cerebral depression. They are met with in the malignant forms of the acute specific fevers, and are of the gravest possible import.

(vi.) *Retraction of the head* is specially characteristic of meningeal inflammations. It is also met with in cases of cervical caries high up, and to some extent in infants with digestive disorders or febrile states.

§ 15. The **Attitude** which is involuntarily assumed by a patient suffering from certain chronic diseases, if he be able to leave his bed, is very characteristic. Thus :—

(i.) In *Paralysis agitans* the head, neck, and thorax are bent



Fig. A.—The attitude typical of PARALYSIS AGITANS; from a plaster cast by M. Paul Richer.



forwards, and the tendency which the patient has to run forwards



is called "festination" (Fig. 4) The disease is recognisable at sight by the smooth expressionless face, fixity of gaze (always looking forwards), the forward bending of the body, and the short steps which the patient takes as he shuffles along.

(ii.) The attitude assumed by children suffering from *post-diphtheritic paralysis* is somewhat similar to the preceding, and is so characteristic that one can often detect the disease as the patient enters the room. The head hangs forward from weakness of the neck muscles, and the "flabbiness" of all the movements is peculiar.

(iii.) The *rigidity of the spine* in rheumatoid arthritis, and in spinal caries, gives a stiffness and awkwardness to all the movements which is very noticeable.

(iv.) Duchenne's *Pseudo-Hypertrophic paralysis* (Fig. 5) is a comparatively rare condition, but the arching forwards of the back, prominence of the buttocks, scapulæ, and calves, and inability to rise from a recumbent posture without the aid of the hands are quite pathognomonic. (See Chapter XVII.)

Fig. 5.—PSEUDO-HYPERTROPHIC PARALYSIS of Duchenne.—This patient is a boy at 9 years under the care of my colleague, Dr. T. Outterson Wood. The illustration shows well the two most characteristic features of the disease—namely, the enlargement of the calves and buttocks, and the arching inwards of the back (lordosis). The disease is often combined, as here, with true muscular atrophy in other parts—e.g., the shoulder girdle, and therefore the scapule project. The child also exhibited the typical manner of getting up from the prone position. The boy was 1 of 4 children, of whom 1 sister died of spina bifida æt. 21 days, 1 brother died of "water on the brain" æt. 6 months, and 1 sister æt. 15 months living and healthy. The patient seemed normal until he began to walk (æt. 2 years), when it was noticed he "lifted his legs too high." The mother was æt. 26 when he was born. No known defect in family history.



## (C.) THE GENERAL CONFORMATION.

§ 16. Under this heading we note (a) whether the patient exhibits any loss of flesh (EMACIATION, *infra*), (b) whether he presents any increase in volume (GENERAL ENLARGEMENT, § 18), or (c) whether he presents any DEFORMITY or DWARFISM (§ 20).

Here we shall meet with several important diseases affecting the skeleton and general growth of the individual, especially Hereditary Syphilis. With the exception of that disease the various causes of such alterations will only be mentioned here. They will be described and differentiated under the Diseases of Extremities (Chapter XVII.), and elsewhere.

VARIATIONS IN HEALTH. The terms Emaciation and General Enlargement of the body are only relative. The healthy man should have an elastic skin, firm muscles, and a slight amount of subcutaneous fat, but *individual variations* are so great that no definite standard can be set up as normal. Health in the wiry nervous man is consistent with a spareness that would indicate disease in his stouter and more phlegmatic brother. The same holds true with regard to *age*. A child has an amount of fatty covering that would be abnormal in adolescence; an old man has atrophy of the soft parts and prominence of the bones which in the middle-aged man could only accompany serious disease. The question of build is very largely one of *heredity*. Stout parents generally have children who tend to become stout, and *vice versa*.

(a) **Emaciation** is necessarily attended by more or less weakness, and the subject is dealt with under General Debility (Chapter XIX.).

The chief causes of Debility with Emaciation are as follows: Malignant Disease, Digestive Disorders and Privation, Diabetes, various Nervous Disorders, Chronic Bright's Disease, Syphilis, Tubercle, and Pancreatic Diseases; and in children, Tabes Mesenterica, Defective Feeding, Diarrhœa, and Hereditary Syphilis.

In *advanced life* the first cause which occurs to our minds, if the patient has lost flesh, is cancer; in *middle age*, diabetes; and in *young adults*, tuberculosis. In tuberculosis of the lungs or elsewhere, emaciation may occur before any physical signs can be detected; indeed, loss of flesh which is accompanied by an intermitting pyrexia generally means latent tuberculosis. In

*infancy* the two most common causes of acute or *rapid* wasting are Defective Feeding and Gastro-intestinal Catarrh. The two most common causes of slow, progressive, or *chronic* wasting in infants are tuberculosis of the intestine and mesenteric glands (Tabes Mesenterica), and Hereditary Syphilis.

§ 17. **Hereditary Syphilis** (also called Congenital Syphilis) has considerable resemblance to the second stage of acquired syphilis (*q.v.*) modified by the fact that it occurs in *INFANCY*. Here the emaciation is very marked, and it is accompanied by other unmistakable signs of the malady. Sometimes the infant is born dead (this being the commonest cause of still-births), but it may be born quite healthy, and remain so for a month or two, then the marasmus (wasting) and other symptoms gradually set in. The face in congenital syphilis has already been described (§ 12 (i.)). “Snuffles,” and mucous patches around the anus, are perhaps the most frequent signs, and these may be followed by various eruptions (see Table below). The numerous symptoms are best given in a tabular form; and this will be the most appropriate place for a description of the succeeding symptoms of hereditary syphilis, which may arise at other times of life; because most of them are external manifestations.

*Treatment of Infantile Syphilis.* The child may die of the marasmus or of some intercurrent complication during the first year of life; and this is happily a not infrequent termination. If he survive the first year there is no danger to life, but he is subject thereafter to a constant succession of the ailments and deformities mentioned in the Table annexed. Much depends on the assiduity of the treatment, which should be continued whether symptoms are present or not, throughout the first two years of life. Mercury may be given by rubbing the Ung. Hyd. into the abdomen, or by wearing a flannel belt smeared with it, or in the form of Hyd.  $\bar{c}$ . *crot.* internally—watching the gums the while. This treatment may affect the growth of both sets of teeth, which become ridged transversely, but no other remedy is as efficacious. Iodide may be given now and then, but it is not so successful until the later manifestations are reached. Cod-liver oil, tonics, and good food are also indicated.

**ADOLESCENT MANIFESTATIONS.** After infancy comes a *latent*

*period* of some years, followed, about puberty, by a fresh set of symptoms (*vide* Table). The LATE MANIFESTATIONS consist chiefly of the results of the infantile and adolescent lesions.

TABLE I.

HEREDITARY SYPHILIS.<sup>1</sup>

## A. INFANTILE MANIFESTATIONS (3 weeks—3 months).

- I. May be born quite healthy. Then symptoms resembling acquired secondary syphilis appear—symmetrical, transitory, etc.
- II. Mucous Membranes { Snuffles.  
Condylomata around anus or mouth.
- III. Marasmus, leading to “senile aspect”; very marked wasting, often fatal.
- IV. Skin { Papular . . . } Always symmetrical, transitory, ham-coloured;  
Scaly . . . } on buttocks because of urine and fæces; in  
Pustular . . . } flexures because of perspiration. Patches of  
Bullous . . . } peeling erythema about face, nates, neck, etc.  
Polymorphic )
- V. Iritis.
- VI. Definite Periostitis—Tenderness of bones and “rheumatic” pains, epiphyseal abscesses, or caries of long bones. Skull—thinning in one place, thickening in another. Skeletal deformities and nodes.

B. ADOLESCENT MANIFESTATIONS (commencing about puberty),  
Which come on after an interval of quiescence of some years, if the child survive the first year of life.

- I. Nebular Keratitis—first one eye, then the other becomes like ground-glass—between 10th and 20th year. Ultimately quite clears up under treatment (Fig. 3).
- II. Deafness—between puberty and 23rd year—comes on with noises in ears, but without pain or otorrhœa—terminates in recovery or complete incurable deafness.
- III. Periostitis of long bones (rarely skull)—generally causes overgrowth sometimes bending, or nodes, occasionally suppuration.
- IV. Synovitis (painless)—knee or other large joint.
- V. Skin, viscera, and nervous system rarely affected at this stage.

<sup>1</sup> This Table is after Fournier, modified.

## C. LATE MANIFESTATIONS (from 15 years upwards).

NOTE.—All of these, being the results of infantile syphilitic inflammations, are absent if syphilitic manifestations have been previously wanting.

- |                         |   |   |
|-------------------------|---|---|
| Constitutional Effects. | { | Infantile build.<br>Retardation of development, of growth, of dentition, of the catamenia.  |
| II. Tegumentary System. | { | Skin—Peribuccal cicatrices radiating from the mouth; Parrot's cicatrices.<br>Eruptions (very rare)—Lupoid ulceration, gradually spreading, may appear.<br>Mucous membranes—Cicatrices of the throat, palate, and round the mouth. Hole in palate, etc.  |
| III. Osseous System.    | { | Cranial malformations—prominent frontaleminences. natiform cranium, asymmetry, hydrocephalus.<br>Nasal malformations—"Duck-nose," depressed septum, "opera-glass nose."<br>Tibial deformities—"Sword-blade" tibia; or curving with shortening; or increased length.<br>Joint lesions—Chronic painless effusions, and distorting arthropathies.    |
| IV. Hutchinson's Triad. | { | 1. Eye { Ocular Malformations.<br>The remnants of interstitial keratitis (striae in cornea), iritis, or choroidal atrophy.<br>2. Ear { Cicatrices of the tympanum, deafness.<br>3. Teeth { Underhung or displaced jaws, irregularities or absence of teeth.<br>Dental Dystrophies—microdontism, amorphism, "pegged teeth" of Hutchinson (Fig. 3). |
| V. Family History.      | { | Miscarriages and still-births in series.<br>Heavy mortality among children in first three months of life.   |

§ 18. General enlargement of the body is much less often met with than diminution. It occurs in *Obesity*, *Generalised Dropsy* (see §§ 9 and 22), *Myxœdema* (see § 9, and under General Debility), and *Acromegaly*.<sup>1</sup> It is probable that the giants of old were specimens of acromegaly. These affections will be described and differentiated elsewhere, but since the treatment of obesity has unfortunately been allowed to get into the hands

<sup>1</sup> I have also met with general enlargement of the extremities in certain rare cases presenting vaso-motor symptoms.

of charlatans and patent medicine vendors, it will be well to add a few remarks on the causes and treatment of that condition.

§ 19. **Obesity** is very largely a question of heredity, and no amount of dieting will then make any difference in some people. In others it is an indication of luxurious or sedentary life, or of indulgence in alcohol. Women frequently become obese just about the menopause. Sometimes it is found in chronic cerebral disease, such as idiocy or tumour. The pathological causes of obesity come under two headings: (i) excessive intake of those foodstuffs known to produce fat (carbohydrates and hydrocarbons); and (ii.) deficient oxygenation. It is probably due to the latter cause that persons with persistent low tension are apt to become fat. Both causes may be in operation. Successful treatment must therefore depend either upon diminution of intake or increase of oxygenation.

**TREATMENT.** Our first duty when consulted about such cases is to examine every organ in the body, especially the heart, lungs, and liver, because excess of subcutaneous fat is often attended by a similar deposit of subpericardial fat; and, if due to alcohol, by fatty degeneration of both heart and liver. Chronic bronchitis and emphysema are also frequently followed by obesity, from deficient oxygenation. If no serious lesion be present there are at least five methods of reduction: (1) To limit the amount of fluid taken with meals. (2) Banting's system consists in excluding all fats, sugars, and starches from the diet, green vegetables and lean meat alone being allowed. (3) Ebstein's system only excludes all sugars and other carbohydrates. (4) Oertel's system is the most complete, and consists in (i.) slowly climbing mountains for several hours daily, inspiring with one step and expiring with the next; (ii.) food mainly nitrogenous, with only small quantities of fats and starches; (iii.) meals taken at regular intervals, and in strictly regulated quantities; (iv.) the fluid limited to  $1\frac{1}{2}$  pints in 24 hours (see also Chapter III.). (5) Some cases of obesity may be successfully reduced by *limiting the food* entirely to *one pound of lean meat* or fish a day, divided into four meals, taken without fluid; between meals the patient drinking as much hot water as possible by constantly sipping it. If the patient will co-operate this method is very successful, but the diet is a Spartan one, nothing but lean meat and hot water being allowed.<sup>1</sup> Thyroid or strychnine are also good in obesity either separately or together.

*Adiposis Dolorosa* is a rare variety of obesity starting with localised deposits of fat, accompanied by tenderness, which gradually become generalised there can be no doubt (the hands, feet, and face escaping), with constant and paroxysmal pains in various situations, great muscular weakness, and partial loss of sensation in certain areas. The disease was first described by Dercum, and since then others have recorded cases, though there are only eight in all, as far as I am aware, all having been females. Dercum, *Internat. Journ. Med. Sci.*, 1892, p. 521 (three cases); Eshner, *Phil. Med. Journ.*, 1898 (one case); Spillen, *Med. News*, 1898 (three cases). A case is figured in the *B. M. J.*, 1909, ii., 1553.

§ 20. **Dwarfism**, or diminished stature, may arise from any cause which affects the growth of the bones of the trunk or limbs, whether local or constitutional. The commonest causes of a stunted condition of the body, in order of frequency, are :—

<sup>1</sup> A case is published in the *Lancet*, 1893, vol. ii., p. 133. There is a soap on the market which is said to contain ox-gall. How it acts is obscure, but of its efficacy in some cases there is no doubt.



(i.) *Rickets*. In this disease there is curving of the long bones together with altered epiphysial growth. This results in "bandy legs," "knock-knee," and other familiar deformities. (See Chapter XV.)

(ii.) *Hereditary Syphilis*, the means of recognising which are fully given above (§ 17).

(iii.) *Curvature of the Spine*, which may take three forms : *kyphosis* (i.e.,



FIG. 6.



FIG. 7.

Fig. 6.—Case of CRETINISM under the care of W. Rushton Parker. The child, *act.* 6, presented the characteristic aspect of a cretin, viz., sunken nose, swollen mouth, small eyes widely apart, coarse hair, and stumpy limbs. Fig. 7. Shows the same child after 6 months' treatment with Thyroid gland, 5 gr. daily.

the convexity projecting backwards), usually due to tuberculous or other disease of the vertebræ; *lordosis* (i.e., a forward projection), usually compensatory, or the result of muscular weakness; and *scoliosis* (a lateral curve). All of these may diminish the stature, but they differ considerably in importance. A certain amount of scoliosis is normal to nearly every one, and the kyphosis of muscular weakness is common enough in old age, as a consequence of which our stature becomes slightly less in advancing years. It is the angular kyphosis which is so serious, as indicating organic diseases of the bodies of the vertebræ.

(iv.) *Cretinism* is a peculiar stunting of the growth which is endemic among children in certain districts, particularly the valleys, of some parts of Switzerland and elsewhere. The appearance is so distinctive that typical cases can be recognised at a distance (Fig. 6). The face is broad and flat, and joined almost without a neck to the body. The skin and hair are coarse, the hands broad and stumpy, the stature stunted, for even when twenty years of age a cretin may be only three feet high. It is due to a perverted or diminished action of the thyroid gland; and when thyroid is administered internally they recover (Fig. 7).

(v.) *Achondroplasia*. A rare condition somewhat resembling and formerly confused with Rickets (see Chapter XVII.).

(vi.) *Osteomalacia*, when this disease involves the spine (Chapter XVII.)

(vii.) In addition to the foregoing there are certain rare conditions, of which the celebrated Tom Thumb and his wife, and the race of Pigmies of Africa met with by H. M. Stanley, are examples, in which the skeleton and the organs are diminished in size, but their proportions maintained. Such cases, however, seem to be functionally normal in every respect.

Some of the diseases above referred to belong so distinctly to the domain of surgery that reference must be made to other works for their differentiation. Others will be described under Diseases of the Extremities.

## CHAPTER III.

### DISEASES OF THE HEART AND PERICARDIUM.

THERE are three noteworthy facts in connection with the diseases of the circulatory system. First, that the l. side of the heart is stronger and much more prone to disease than the r. side ; secondly, that the arteries are, in a corresponding manner, much thicker and more often diseased than the veins ; and thirdly, the very prominent part played by heredity in chronic disorders of the heart and arteries.

The saying of Björnson that "Heredity is a condition. not a destiny,"<sup>1</sup> applies here ; for although its application is chiefly ethical, it may also be employed in a physical sense also, as is witnessed by the fact that careful living may do much to counteract the hereditary tendency to early death from chronic cardio-vascular disease.

Following out the plan adopted in this work, we shall consider :—

First, the SYMPTOMS which lead us to infer the presence of cardiac disease.

Secondly, the PHYSICAL EXAMINATION of the patient ; and

Thirdly, the DIFFERENTIATION OF THE VARIOUS DISEASES which affect the heart and pericardium, their diagnosis, prognosis and treatment.

#### PART A. SYMPTOMATOLOGY.

The general symptoms (*e.g.*, breathlessness, dropsy, etc.) of cardiac disease, as distinct from the local signs referable to the heart, should be studied very carefully, inasmuch as the gravity of any given case depends not so much on the local signs present as on the general condition of the patient.

The **THREE CARDINAL SYMPTOMS** of diseases of the heart and pericardium are **Breathlessness, Dropsy, and Cyanosis.** To these may be added **Palpitation, Præcordial Pain, Syncope,**

<sup>1</sup> "The Heritage of the Kurts."

sometimes **Cough**, and, in acute affections, **Pyrexia** and its concomitant symptoms. **Sudden Death** is more frequent in disorders of the heart than in disease of any other viscus, and it is sometimes unattended by any previous manifestation of heart disease.

§ 21. **Breathlessness**, or *Dyspnœa*, is a very constant symptom in all diseases in which the heart is unequal to the work demanded of it. Breathlessness may be present without cardiac disease; but it may be affirmed that no serious affection of the **CARDIAC WALL** can exist without some degree of breathlessness. It may be only on exertion, such as walking up a few stairs, but it can always be elicited in some degree.

Marked disease of the **VALVES** of the heart may, however, exist for many years—provided the obstruction so caused is adequately compensated for by increased growth in its muscular wall—without the patient having any noteworthy symptoms, or even being aware of its existence, until the cardiac wall begins to degenerate and its cavity to dilate. Then, as time goes on, breathlessness is sure to appear, and it is for this symptom that you are mostly consulted in heart cases. In the later stages it becomes so pronounced that the patient is unable to breathe lying down, and night after night is passed sitting upright in a chair, or at best, propped up with pillows in bed (*orthopnœa*).<sup>1</sup> Towards the end, in a certain number of cases, a larger proportion in my experience than is generally supposed, some degree of *Cheyne-Stokes respiration* may be observed.

**CAUSES OF BREATHLESSNESS (DYSPNŒA).** Difficult breathing may arise under five different groups of disorders.<sup>2</sup>

1. **Cardiac disease.** The *dyspnœa* of heart disease has no intrinsic features which distinguish it from that due to other causes, excepting the fact that it is apt in some cases to be paroxysmal. There is, however, usually a history, or evidence, of some of the other symptoms of cardiac disorder. In cardiac valvular disease the amount of breathlessness present and the distance a patient can walk without producing it are, of all symptoms,

<sup>1</sup> This word simply implies very urgent *dyspnœa*—*ὀρθρος*, Greek, erect.

<sup>2</sup> This does not include the *dyspnœa* associated with vaso-motor spasm or with vaso-motor paresis and states of low arterial tension, such as may be met with in great prostration or bodily fatigue. This kind of *dyspnœa*, I have observed, has for its chief feature a sighing character of the respiration, and long-drawn sighs occur every few seconds.

the most valuable indications as to the amount of degeneration of the heart-wall (cardiac failure) present in any particular case.

2. **Pressure of surrounding organs on the heart**, such as a dilated stomach, ascites, mediastinal tumours, or obesity. Murchison used to teach that dyspnœa and nearly all the symptoms of cardiac disease (excepting cyanosis) may be produced by dyspepsia without any structural disease of the heart.

3. **Laryngeal or tracheal obstruction.**

4. **Pulmonary disease**, of which Emphysema is the most common.

5. **Toxic or hæmic conditions**, the most frequent of which is certainly anæmia. Deficient aëration of the blood, poisonous conditions of the blood, such as uræmia (Chronic Bright's Disease), diabetes, and all pyrexial states, may be attended by dyspnœa, caused in this way.

*Causes of Breathlessness which are apt to be overlooked.*—The differentiation of the various forms of cardiac disease will be given in the following pages; but, supposing a patient over 35 or 40, who complains of breathlessness, presents no definite signs of cardiac or pulmonary disease, nor any evidences of dyspepsia or anæmia, then there are certain conditions which should be suspected:—

1. **Cardiac Enfeeblement or Fatty Degeneration**, in which case the sounds and impulse would be very feeble, and the other signs mentioned below (§ 55) should be present.

2. **Arterial Sclerosis**, in which case the radials would be hard and cord-like, but smooth, unless atheroma were also present: and the arterial tension high (excepting in the very last stage of the disease). Here also there would be giddiness, especially on assuming the erect posture, and the other symptoms mentioned below (see § 70).

3. **Aortic Stenosis**, the murmur of which is sometimes very difficult to detect, especially when attended by cardiac enfeeblement.

4. Deep-seated **Aneurysm of the aorta** and other **intra-thoracic tumours** may give rise to the breathlessness and general symptoms of heart disease without the physical signs. In such cases the dyspnœa may be paroxysmal.

5. **Coronary Obstruction** (*i.e.*, diminution of the calibre of the coronary arteries by atheroma, calcification, or other disease). In this obscure condition, the patient probably complains also of "dizziness in the head" on suddenly assuming the erect position, from the incapacity of the heart to pump the blood to the head. But this condition can never be more than suspected during life.

In a patient under 35 or 40 the three following causes of UNEXPLAINED BREATHLESSNESS may be suspected:—

6. **Cardiac Syphilis** may be unattended by any signs or symptoms, excepting breathlessness (§ 55). Happily the condition is very rare.

7. **Adherent Pericardium**, also, is often unattended by any physical signs (§ 41).



8. **Latent Pulmonary Disease**, and especially latent pulmonary tuberculosis, should always be suspected in cases of breathlessness without obvious cause.

9. When severe dyspnœa sets in suddenly in the course of cardiac or acute renal disease, or during an attack of *scarlatina*, the chest should always be carefully examined, because double hydrothorax may set in rapidly without any general dropsy or other warning symptom; as in a case mentioned by Osler, and a similar one which I have had the opportunity of observing.

§ 21a. **Paroxysmal Dyspnœa** is that form of dyspnœa which occurs in attacks from time to time without apparent reason. It is apt, as above mentioned, to occur in some cases of cardiac disease, and in any given case our attention should first be directed to the heart. But there are several other conditions which one would suspect in a patient in whom the chief or only symptom consists of paroxysms of breathlessness.

(1) **ANEURYSM** and other **INTRA-THORACIC TUMOURS** may give rise to paroxysmal dyspnœa before other signs can be made out.

(2) In **ASTHMA** the attacks of breathlessness are typically paroxysmal: and if the disease is of long standing it is usually associated with alterations in the shape of the chest.

(3) Paroxysms of dyspnœa occurring at night is often one of the first symptoms of **CHRONIC BRIGHT'S DISEASE** (see general debility), and is spoken of by the patient as asthma.

(4) **NEUROTIC dyspnœa**. Some neurotic patients are liable to attacks or paroxysms of panting respiration resembling the panting of fear. It usually ceases when the patient converses with you, and is, of course, unattended by any signs in the lungs.

(5) **LINGUAL VARIX**—i.e., a varicose condition of the veins at the root of the tongue—may give rise to severe paroxysms of dyspnœa.<sup>1</sup>

(6) **FOREIGN BODIES** in the trachea in children, and polypi or papillomata of the larynx in adults, give rise to paroxysms of dyspnœa.

(7) Sudden dyspnœa, coming on during vomiting, is the main indication of that rare accident, **RUPTURE OF THE ŒSOPHAGUS**. This dyspnœa is due to pneumothorax, by and promptly opening the thorax on that side. the patient's life might perhaps be saved.<sup>2</sup>

§ 21b. **Cheyne-Stokes' Respiration** (so called after its first observers) consists in its typical form of a series of eight or ten rapid inspirations, gradually increasing in depth and rapidity and then gradually dying away, each series being separated by a pause of five to thirty seconds (the stage of apnœa), in which there is hardly any respiratory movement (Fig. 8).

In a modified form, without the apnœa pause, Cheyne-Stokes' breathing is by no means infrequent. It seems to indicate a want of harmony between the cardiac and the vaso-motor regulator mechanism. It is a very serious symptom, and it appeared in the great majority of those of my cardiac patients in the infirmary who were closely observed *towards the end of life*. Its principal causes are as follows:—

<sup>1</sup> A case of paroxysmal dyspnœa cured by the removal of a lingual varix is published by M. P. Mayo Collier in the West Lond. Med. Chir. Soc. Trans. 1897, p. 206.

<sup>2</sup> A few cases of this accident have been recorded. If it were recognised the thorax might be opened without fear on the side of the pneumothorax, and the œsophagus stitched. See Discussion at Roy. Med. Chir. Soc. in spring of 1900.

- (1) CARDIAC DISEASE, which is certainly its commonest cause.
- (2) URÆMIA.
- (3) APOPLEXY.
- (4) TUBERCULAR MENINGITIS, and other states of cerebral congestion.
- (5) ARTERIAL SCLEROSIS (used in its widest sense).
- (6) SUNSTROKE.

Thus it will be seen that the three pathological conditions in which it is apt to occur are—states of cardiac failure, states of cerebral congestion, and toxic blood conditions. The patient seldom lives longer than a few days with such a condition, but a remarkable exception to this occurred under



FIG. 8.—CHEYNE-STOKES' RESPIRATION.

Respiratory tracing of Cheyne-Stokes' breathing, for which the author is indebted to Dr. C. O. Hawthorne, who took the tracing from a case of cerebral embolism under his care in the Western Infirmary, Glasgow. Dr. W. S. Cook, the House Physician, kindly supplied the notes of the case, which is one of considerable interest.

*Summary.*—"The patient, a man aged 54, was admitted May 24th, and died June 4th, 1895. He had been attacked suddenly, eleven days before death, with hemiplegia and quasi-aphasic phenomena, attended with marked Cheyne-Stokes' respiration and loss of control over bladder and rectum. There was a history of two previous 'strokes,' in which the speech was suddenly affected, but no accompanying paralysis recognised; recovery regarded as complete after each stroke. During his residence in the infirmary he had several 'attacks' resembling rigors, with marked acro-asphyxia in the upper limbs; respiration at those times becoming regular though somewhat stertorous. Sudden unconsciousness and death followed one of these attacks. Temperature subnormal, except in one rigor it was 100° F. about twelve hours before death. Urine in one specimen showed slight albumen. The interesting point about this case was the well-marked and long-sustained Cheyne-Stokes' respiration. He walked into the receiving room and upstairs with this peculiarity in the respiration, and it lasted for over a week almost continuously. The apnoea pause varied in this case from 15 to 65 seconds, and as many as 25 and 28 respirations were counted in the intervening cycles. It was a curious fact that while the Cheyne-Stokes' breathing continued, there never was any sign of cyanosis or lividity anywhere; and that when the cyanosis became apparent in the arms, the breathing was regular though stertorous. The lividity in the arms at this time was sometimes extreme, like a plum; and there were scattered through it bright red spots like those seen in Raynaud's disease." [This alternation of the cyanosis with the Cheyne-Stokes' breathing tends to support the view that this peculiar alteration of the respiratory rhythm is due to a want of harmony between the cardiac nervous apparatus and the vaso-motor regulator mechanism.]

"At the post-mortem examination thrombi were found in the heart, and evidence of an old softening in the left side of the brain, involving Broca's convolution. It involved a very large area, which was flattened and collapsed. On the right side there was a recent softening, with a plug in the artery, involving the island of Reil and surrounding parts, including a part of the internal capsule."

my notice in a case of focal lesion of the pons, producing crossed hemiplegia, in which it persisted, with only occasional intermissions, during the last six months of the patient's life; another exception is seen in the case from which the illustration is taken.

§ 22. **Dropsy** is a chronic effusion of fluid (serum) into the subcutaneous tissue (when it is known as anasarca) or into the serous cavities (as in hydrothorax, hydropericardium, ascites). The

former, **anasarca**, is the variety of dropsy we are now concerned with, for it is a very constant feature of some forms of cardiac disease. General anasarca has to be differentiated from myxœdema, in which the swelling is harder, and does not pit on pressure by the finger, as anasarca does. It is best to apply the pressure over a bone, such as the lower end of tibia on its inner aspect.

*Causes.* The causes of localised dropsies are given in "Diseases of the Extremities." There are *three varieties of general anasarca*, which differ from each other both pathologically in their origin, and clinically in the course which they pursue.

1. **Cardiac Dropsy** (1) *starts*, and throughout the case predominates, in the *most dependent parts*, that is to say, in the legs if the patient has been walking about, or in the back if he has been lying in bed. On inquiry, the patient may complain that the ankles swell towards evening around the top of the boot, and on pressing over the tibia the dent will remain after the removal of the finger. (2) Other signs and symptoms of enfeeblement or dilatation of the cardiac wall are present; and perhaps those of valvular disease as well. (3) In the history of the case dyspnœa will have *preceded* the dropsy.

Dropsy does not occur with equal frequency in all forms of cardiac valvular disease. It is common in disease of the mitral valves, but rare in aortic disease, at any rate until quite the end. The dropsy which complicates pulmonary disease has the same features as cardiac dropsy, because it is the resulting cardiac dilatation which produces the dropsy.

2. **Hepatic Dropsy** (1) always begins and predominates *in the abdomen* (ascites), although the legs may swell subsequently by reason of the pressure of the fluid on the veins within the abdominal cavity. (2) There may be also enlargement and other signs of the liver affection which has given rise to the condition; and if these be absent some other cause of obstruction to the portal vein should be sought (Chapter XIII.). (3) The dyspnœa will have *followed* the abdominal enlargement.

3. **Renal Dropsy** is (1) *general in its distribution* from the beginning, occurring in the legs and eyelids at the same time; though it is probable that the œdema round the eyes on rising in the morning first attracts the attention of the patient or his friends.

(2) Examination of the urine reveals the features of renal disease (Chapter XII.). (3) The patient presents a characteristic pale or waxy appearance.

In some cases of general anasarca associated with albuminuria, the question arises whether the dropsy is of renal or cardiac origin? This question may be answered by finding the liver enlarged, for this is a natural sequence of cardiac valvular disease, but not of renal disease.

*Prognosis.*—The dropsy of cardiac disease admits of a mechanical explanation,<sup>1</sup> being due to the backward pressure within the veins. Its occurrence is therefore an indication and a measure of the amount of obstruction to the circulation on the right side of the heart.

The *treatment* of all forms of dropsy should be directed to the removal of the cause. But even if this be not removable, the dropsy may frequently be alleviated. The limbs should always be rested, raised to the same level as the body, and kept warm. The additional support of a well-adjusted flannel or stocking bandage is a great comfort to the patient, and helps to prevent further effusion. Diuretics and diaphoretics should be employed. These failing, we may (a) employ Southey's trocars, the patient being wrapped in blankets and the fluid allowed to slowly drain away; or (b) make punctures in the skin of the limb with a small two-edged scalpel. Six or eight punctures are sufficient, the positions of the veins being avoided. These procedures are not without some danger of erysipelas and cellulitis, and strict antiseptic precautions should be followed. Dropsical limbs have a tendency to the development of Eczema, Erythema, Cellulitis, and Epidemic Exfoliative Dermatitis, and our efforts must be directed to prevent the supervention of such complications.

OBSCURE CAUSES OF GENERAL ANASARCA. If, in a patient who complains of dropsy, no marked evidences of cardiac, renal, or hepatic disease are discoverable, the following causes may be *suspected* :—

1. **Anæmia** is not unfrequently attended by some swelling of the ankles at the end of the day. This may appear quite early in chlorosis, but is rare in pernicious anæmia.

2. Other **defective blood conditions**, such as leucocythæmia, may be accompanied by dropsy. Swelling of the feet and ankles may be

<sup>1</sup> That this is not the only cause is evidenced by certain facts. Thus, occasionally, cases of mitral disease are unaccompanied by dropsy at any time. And again, the degree of cyanosis, which is sometimes also a measure of venous stagnation, may have no ratio to the amount of dropsy present.



present in the last stages of many exhausting diseases, such as phthisis, in septic states, and in cases of insufficient nutrition and old age.

3. In **Fatty heart** anasarca is not a prominent symptom, but a slight degree is frequently present.

4. Among the less frequent causes of dropsy, rarely seen in this country, are **Beri-Beri** and **Epidemic Dropsy**. In Beri-Beri there are symptoms also of peripheral neuritis. Epidemic Dropsy is the name applied to an outbreak which occurred in Calcutta in 1878-80, in which the leading symptom was anasarca, beginning in the lower extremities, accompanied by moderate pyrexia, gastro-intestinal disturbance, and sometimes itching, urticaria, or other rashes.<sup>1</sup>

§ 23. **Palpitation** is the sensation of "fluttering in the chest" experienced by a person when he is conscious of the beating of his heart. It arises under two sets of conditions. It is said to be **SYMPTOMATIC** when a cause can be assigned to it, such as heart disease, anæmia, intra-thoracic tumour, etc. It is said to be **IDIOPATHIC** or **functional** when no organic cause can be discovered to account for the symptom. (Compare also Paroxysmal Tachycardia, § 43.)

**SYMPTOMATIC PALPITATION** may arise from (a) causes referable to the heart itself (causes 1 to 3); or (b) morbid conditions outside the heart (causes 4 to 9).

(1) Most **structural diseases of the heart**, especially such as are attended by rapidity and irregularity of its rhythm, are attended by palpitation. In the case of patients who consult us for this symptom our attention therefore should first of all be directed to a thorough examination of the heart and pericardium.

(2) Palpitation is also the leading indication of the **irritable heart**, in which there may be no discoverable structural disease. Irritable heart is found in young men who have overtaxed their strength in athletics or military work, and occasionally in young women. Palpitation is its most obvious symptom, with a very quick pulse, or one which *easily becomes rapid*, and sometimes with high arterial tension. Breathlessness, sleeplessness, incapacity for prolonged exertion, and nervousness often accompany the palpitation. Hypertrophy is a common result; and unless rest be ordered the heart will become dilated. Complete rest is the chief indication

<sup>1</sup> Trans. Epidem. Soc. Lond., vol. xii., p. 55 (New Series).



for treatment, and is generally successful. Irritable heart is in my belief in some cases a variety of neurasthenia (*q. v.*).

(3) Various **nervous causes**, such as fright, fear, or other emotion, especially after an exhausting illness, give rise to palpitation. (Compare Paroxysmal Tachycardia, § 43.)

4. In **anæmia** the palpitation is a very constant and often a distressing feature.

5. In **dyspepsia**, palpitation is very often present. In such cases it frequently occurs at night, especially after taking a heavy meal. It may, in these circumstances, be accompanied by morbid dreads, *e.g.*, of impending death, by breathlessness, cardiac pain, and by other cardiac symptoms.

6. Certain **local conditions**, such as thoracic or abdominal tumour, or dilated stomach, which hamper the heart's action, may produce palpitation although the heart be healthy.

7. The excessive use of **certain drugs** or **articles of diet**, notably tobacco, tea, coffee, and alcohol.

8. In **Graves' disease** (exophthalmic goitre) violent palpitation and greatly increased rate of the heart are prominent features. In quite a number of my cases this and the other nervous symptoms of the disorder had existed for many months, or years, before the two diagnostic features—thyroid enlargement and exophthalmos—became obvious. Graves' disease should always be suspected in cases of persistent palpitation for which no cause can be made out.

9. In **hysterical** subjects, palpitation is a symptom often complained of, and occasionally it takes the form of a definite and somewhat alarming attack (see a case § 43).

§ 24. **Pain in the chest** is not always present, even in grave cardiac disease. A feeling of discomfort or constriction, or a sense of suffocation, is a symptom frequently present when the action of the heart is deranged by functional or structural diseases—oftener perhaps in functional.

To avoid certain *fallacies*, ascertain if there be tenderness on pressure. If so, the lesion is probably a *neuralgia* of the intercostal nerves; or the *inframammary pain* so common in hysteria, which may sometimes be distinguished in this way from pain of cardiac origin. Occasionally this latter can only be distinguished by the presence of hysterical stigmata.

There are **FOUR GROUPS OF CAUSES** under which præcordial pain may arise:—

(a) When præcordial pain occurs as the result of **structural disease of the heart** (due to overstrain), it usually consists of a diffuse dull aching, most severe at the apex. As in other organs having a serous covering, pain is more often present when

that covering is inflamed (pericarditis) than when the substance of the organ is affected. But pain may be altogether absent; and it is surprising what serious valvular derangement of the heart may exist without the occurrence of any pain at all.

(b) **Cardiac pain of functional origin** may be due to (i.) *pressure upon the heart* by a distended stomach or abdomen; the differential features of this pain are—it is greatest at the base of the heart, aggravated by the recumbent posture, and associated with dyspnœa. (ii.) *Reflex Pain*, may be referred from stomach (mostly) or uterine disorders (occasionally). (iii.) *Nervous Pain* due to profound grief, sudden fright, or other violent emotion, is of a sharp character, referred to the præcordium.

(c) Various **organic affections outside the heart and pericardium** may give rise to præcordial pain; thus we may have *intercostal neuralgia*, especially the neuralgia which precedes and follows herpes zoster; *intercostal rheumatism*, and various *pleuritic affections*. Pain in the chest is also present in *spinal caries* and *carcinoma of the vertebra*.

(d) **Angina Pectoris** is a disease manifested by paroxysmal attacks of extremely severe constricting pain in the chest, with a sense of suffocation and other symptoms. It will be referred to under the acute disorders of the heart (§ 44).

But, in cases of unexplained pain in the chest, and in the absence of cardiac signs, *mediastinal tumour* or *aneurysm of the aorta*, either of the arch or of the descending aorta,<sup>1</sup> should always be suspected.

In the *treatment* of præcordial pain an endeavour should be made to ascertain and relieve the cause, but much relief may be obtained temporarily by the application of an opium or belladonna plaster, belladonna liniment, or glycerine of belladonna.

§ 25. **Syncope** is suspended animation due to anæmia of the brain. It is often preceded by giddiness, nausea, and a feeling of faintness. The face is ashy pale and the pulse and respiration feeble. Its advent is usually sudden, but recovery, after the attack has lasted some minutes, is gradual.

<sup>1</sup> In a case of aneurysm of the descending thoracic aorta which I have recently seen, almost the only symptom or sign, besides breathlessness, during eighteen months—up to the time of sudden death from rupture of the aneurysm into a bronchus—was continuous pain in the præcordial region. It extended round from the back on the left side, and was thought to be intercostal neuralgia.

*Diagnosis.* Syncope has to be distinguished from *epilepsy minor*, which it resembles in many respects. First, epilepsy minor (petit mal) is usually preceded by an aura, though this is evident to the patient only; secondly, its advent is more sudden than syncope, and the return to consciousness equally sudden and complete, for the patient in petit mal can go on with his usual avocations immediately afterwards. Thirdly, syncope rarely occurs without some definite determining cause, although it may be of a trivial nature; such, for example, as a heated room, or the sight of blood. Finally, in epilepsy minor there is generally a history of major attacks at some time.

*Causes.* Syncope nearly always arises from either structural or functional derangement of the heart; more frequently the latter. A careful examination of the heart should always be made, because, as an indication of STRUCTURAL DISEASE, syncope is a symptom of considerable gravity; whereas the nervous faints of FUNCTIONAL DERANGEMENT are of comparatively little moment.

(a) The **nervous faints** due to functional derangement of the heart are happily the more frequent. They occur chiefly in young, anæmic, and nervous females; who, when exposed to grief, bereavement, or any sudden emotion, or too hot rooms full of vitiated air, develop the familiar "fainting attack." Slight transient syncopal attacks are indeed one of the "stigmata" of the hysterical diathesis, the other three being hemianæsthesia, ovarian tenderness, and globus.

(b) As a symptom of **structural heart disease**, syncope is a much more serious matter. It is a not infrequent symptom in any form of cardiac disease attended by enfeeblement of the heart's action, and is serious as indicating weakness of the *cardiac wall*. It is more often met with in aortic than in mitral valve disease. It may be the first and only symptom of fatty (§ 55) fibroid or other degeneration of the heart. Syncopal attacks, preceded by giddiness, may arise in old people who are the subjects of arterial thickening and degeneration; this being the cause of what is known as "senile syncope." So important is it to distinguish between the two kinds of fainting attacks that their differential features are given in a table. In both there is pallor of the surface and there may be feeble pulse, though the

pulse in nervous faints is sometimes unaltered. Both recover best in the recumbent position.

**Cardiac syncope**, associated with structural derangement of the heart.

Usually adults; both sexes equally affected.

May come on without any apparent cause, or after excessive exertion.

Not accompanied by emotional manifestations.

May be fatal.

Evidences of cardio-vascular degeneration and its causes.

If no symptoms suspect fatty heart.

**Nervous faints**, in which only the nervous apparatus of the cardio-vascular system is deranged.

Females, young or at menopause.

Some determining cause always present (*e.g.*, emotion), acting on the nervous system.

Often preceded or followed by crying or laughter and other emotional symptoms.

Never fatal.

Sometimes other evidences of the hysterical diathesis—*e.g.*, hemianæsthesia, ovarian tenderness, globus.

*Obscure Causes.* In any case of syncope in which the cause is not sufficiently obvious, the following conditions may be passed in review:—

(a) *Acting directly through the heart*:—(i.) Latent organic disease of the heart, such as fatty degeneration, which should always be suspected in obscure cases; (ii.) Compression of the heart, as by corsets or by mediastinal tumour; (iii.) Profuse internal hæmorrhage; (iv.) Drugs and asthenic poisons acting on the heart.

(b) *Acting through the nervous system*:—(i.) Emotions and fatigue; (ii.) Excessive heat, as in hot rooms or Turkish baths; (iii.) Violent injury or operation; (iv.) Irritant poisons, or injury to the intestines.

(c) *Acting through the blood and blood vessels*:—(i.) Anæmia, debility, hunger; (ii.) increased peripheral resistance in the arteries with insufficient cardiac hypertrophy; (iii.) suddenly assuming the erect posture, as in jumping from bed, may produce syncope in the aged; (iv.) sometimes, in addition to the preceding, the splanchnic veins are suddenly dilated by emptying the bladder, and this leads to anæmia of the brain and syncope.

*Prognosis.* Syncope in the young is, as we have seen, usually a neurosis, whereas in the aged it generally means cardio-vascular degeneration. In the former therefore it is usually as trivial as in the latter it is serious—the gravity depending upon the nature of the lesion.

*Treatment* (see Causes above). Place the patient immediately in a horizontal position, with the head low. This may be most readily done on the floor, but in a crowded audience instruct the patient to bend forward and lower the head between the knees. Apply ammonia to the nostrils, throw cold water on the face, and

in severe cases apply a mustard plaster over the heart. If recovery does not promptly take place and the pulse be very feeble, a hypodermic injection of 15 or 20 m. of ether or brandy, or 3 or 4 m. of liq. strychninæ, may be resorted to. For further treatment see Collapse (Chap. XIX.).

§ 26. **Cough** is a symptom which belongs more especially to diseases of the lungs (Chapter VI.), but it is met with in diseases of the heart under two circumstances. First, the lungs are very often involved secondarily to the heart, especially when the right side is diseased, and then the patient has the cough usual to pulmonary disorders. Secondly, when the aorta by its enlargement presses on the trachea, or on the recurrent laryngeal nerve, a peculiar dry, brassy, or, as it is aptly called, the "gander" cough is present, which is so characteristic as to be in itself a diagnostic feature of aneurysm of some considerable value.

§ 27. **Cyanosis** (κυάνεος, blue) is the lividity of the surface which appears when the skin becomes congested or the blood too purple or venous in character. It is not one of the most common symptoms in heart disease, but it is one of the most serious and unmistakable evidences of enfeebled or retarded circulation. It is generally most pronounced on the lips, fingers, and toes, and the skin may vary in colour from a faint purple almost to a black. When only a slight degree of cyanosis is present, it may be detected by closely examining the roots of the nails. Cyanosis is an indication of either:—

1. Admixture of the arterial and venous blood-currents (*e.g.*, Congenital Heart Disease<sup>1</sup>), or—
 

{	<ol style="list-style-type: none"> <li><i>a.</i> venous stasis, a slowing of the blood-current (<i>e.g.</i>, that occurring in the later stages of some cardiac diseases).</li> <li><i>b.</i> defective oxygenation in the lungs (<i>e.g.</i>, that occurring near the end of some pulmonary diseases).</li> </ol>
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2. Deficient aëration.

Sometimes both *a* and *b* are in operation together; but the lividity of cold, rigors, and collapse (*e.g.*, cholera) are probably

<sup>1</sup> Many observers now hold that admixture of blood-currents is not a factor in cyanosis even in Congenital Heart Disease. Undoubtedly the venous stasis, and deficient aëration of the blood which occurs in this disease, are very nearly, if not quite sufficient to account for the cyanotic condition.



instances of venous stasis alone. Increased viscosity of the blood is the proximate cause of the latter.

1. If the patient whose leading symptom is cyanosis be a child who has presented this lividity for a long time, perhaps throughout life, the disease is almost certainly **Congenital heart disease** though this is not a very common disease. The lividity may be very marked without, strange to say, the child appearing to suffer any very great inconvenience.

2. The presence of cyanosis in adult life, forms indisputable evidence, if angio-neurotic cyanosis be excluded, of organic disease of the **heart** (especially the right side), or of the **lungs**, or **both**. It is generally a serious and it may be a lethal indication.

The *Etiology of Cyanosis*—which depends for the most part on defective oxygenation of the blood—may be arranged into ( $\alpha$ ) those in which the onset of cyanosis is usually GRADUAL, and ( $\beta$ ) those in which the onset is usually SUDDEN. *In general terms, the sudden occurrence of cyanosis in those conditions in which its advent is usually gradual is a prognostic indication of considerable gravity.*

( $\alpha$ ) Causes of cyanosis in which the onset is usually GRADUAL :—

1. *Chronic bronchitis* with *Emphysema* is perhaps the commonest cause of cyanosis. It comes on quite gradually, and slowly increases ; but any sudden increase in the degree of cyanosis is of very grave import.

2. In *mitral* valvular disease more or less cyanosis is generally present ; but never in uncomplicated *aortic* disease. So constant is this, that it aids us considerably in deciding which valve is affected.

3. In primary disease of the *cardiac wall* cyanosis, if it does occur, is a symptom of considerable gravity.

4. Gradual compression of the lung by a large amount of *pleuritic effusion* prevents due expansion of the lungs, and therefore the aëration of the blood.

5. Gradual compression of the *bronchi* by enlarged glands, aneurysm, or other intra-thoracic tumour has the same effect.

6. *Acute miliary tuberculosis* of the lungs, or any other chronic infiltration, as by cancer, diminishes the aërating surface of the lungs.

7. The presence of large *abdominal tumours* forcing up the diaphragm prevents full expansion of the lungs, and consequently cyanosis.

8. *New growths* in the bronchi, such as cancer ; or in the larynx, such as polypi, prevent the due entrance of air into the lungs.

( $\beta$ ) The most common causes of cyanosis which comes on SUDDENLY are : —

1. An extensive pneumonia, especially when supervening upon emphysema ; or acute bronchitis, especially in children. The cyanosis which accompanies an extensive pneumonia is due not so much to the fact that a large part of the respiratory area is rendered useless as that the healthy area does not have time to expand and take on the additional work. In more chronic diseases, such as phthisis, a much larger extent of lung may be diseased without any appearance of cyanosis.

2. Diphtheritic membrane invading the air passages.

3. Spasmodic closure of the glottis (spasmodic croup).

4. Foreign body in the larynx, a condition which should always be borne in mind when cyanosis supervenes suddenly in children.

5. Oedema of the glottis.

6. Pressure on the trachea by an enlarged thyroid, etc.

7. Pneumothorax.

8. Paralysis of the respiratory muscles.

9. Paralysis of the abductor muscles of the vocal cords acts similarly ; as in a case which I have recently seen in a patient suffering from *Tubes Dorsalis*. The laryngeal crisis took this form, and was attended by considerable danger to life.

The *Treatment* must have reference to the cause; but in several cardiac conditions cyanosis, if unaccompanied by dropsy, is a distinct indication for venesection. But when marked anasarca is present, it indicates generally that the venous stasis is too great to admit of relief by this measure.

§ 28. A **Sallow hue** of the skin is characteristic of *aortic* valvular disease, which in this respect presents a marked contrast to the plethoric florid appearance of patients with *mitral* valvular disease. This sallowness is distinguished from jaundice by the absence of the yellow colour from the eyeballs and the absence of bile in the urine. True jaundice, however, does arise in cardiac disease as a symptom of the hepatic congestion, which is met with more often in mitral than in aortic disease.

§ 29. **Pyrexia** and its concomitant symptoms (see Chapter XVIII.) are present in most of the *acute disorders* of heart and pericardium. The temperature in Endocarditis has a tendency to be of an intermittent or remittent type, with an irregular range, such as that met with in septic diseases.

§ 30. **Sudden death**, or death, say, within a few hours of the apparent commencement of the illness, is a frequent mode of termination of disease of the heart, and it may be the first symptom of disease of this organ. The chief conditions under which sudden death occurs are as follows. The first six of these have reference to the cardio-vascular system.

1. Among the various forms of *cardiac valvular disease* sudden death is more frequent in aortic than in mitral disease. But sudden death, and, in general terms, the prognosis, depend more upon the condition of the wall than of the valves.

2. It is liable to occur in all forms of primary disease of the *cardiac wall*—*e.g.*, fatty and syphilitic heart (§ 55).

3. It is a very common termination to *aortic aneurysm* (§ 56).

4. A patient may die with the first attack of *Angina Pectoris*.

5. Sudden profuse *hæmorrhage* internal or external.

6. Pulmonary *embolism*, *e.g.*, from air in the veins, clotting in the heart (as in the performance of transfusion).

7. The conditions which cause *Coma* may also result in death which is relatively sudden (Chapter XVI.).

8. Nerve diseases which in their progress involve the *medulla*,

terminate suddenly; and, thus, among the rarer causes, atlanto-axoid disease and syringomyelia may be mentioned.

9. Sudden emotion, injuries to the head, and other diseases acting on the *nervous system* by Shock (Chapter XIX.).

10. Suddenly acting *poisons*, such as prussic acid, a large dose of morphia or carbolic acid, aconite, veratria, etc.

11. Sudden rupture of a large cyst, an internal organ, or other cause of *Collapse* (Chapter XIX.).

12. Foreign bodies in the trachea, or other causes suddenly stopping the respiration (*asphyxia*).

#### B. PHYSICAL EXAMINATION.

§ 31. **Landmarks of the chest.** There is a raised *ridge* on the sternum formed between the manubrium and gladiolus which can always be felt, opposite the 2nd costal cartilage (c.c.); and the other ribs can be counted from the second one. The *nipple* is usually situated just external to the 4th c.c., near its junction with the rib; it should correspond to a vertical line dropped from the middle of the clavicle. At the back, the *lower angle* of the scapula is near the 7th rib; and the *scapular line* is a vertical line drawn through the inferior angle of the scapula. The position and relations of the heart can be studied in Fig. 11, which is a sketch taken from the cadaver. The various regions of the thorax named for convenience of reference are given in Fig. 35 in the chapter on pulmonary diseases. The ordinary methods employed for the physical examination of the heart, and the pericardial sac within which it is suspended by its *attachments at the top*, are INSPECTION, PALPATION, PERCUSSION, and AUSCULTATION. Modern surgical methods enable us occasionally to employ the method of ASPIRATION under due precautions; and RADIOGRAPHY is coming into vogue.

§ 32. **Inspection.** Note, *first*, the attitude of the patient, and the amount of movement he can make without becoming *breathless*. Note also if there be any visible pulsation of the arteries and veins at the root of his neck. In some cases it is desirable to take a side view, and slight deviations from the normal can often be better observed by looking over the patient's shoulder.

*Secondly*, most useful information may be obtained by simply observing the aspect and appearance of the patient, for the pallor

or sallowness of the skin in cases of *aortic* valvular disease presents so marked a contrast to the florid cheeks of *mitral* disease as to form a most valuable aid to diagnosis. Notice also if the dropsy of the ankles or back, which sooner or later attends mitral disease, is present.

*Thirdly*, carefully observe that part of the chest wall which lies in front of the heart. It is important to notice if there be any undue pulsation or bulging in any part of the chest. The forcible displaced apex-beat of cardiac hypertrophy; the wavy, diffuse pulsation of cardiac dilatation and pericardial effusion; the epigastric pulsation and throbbing jugular veins in cases of dilated r. ventricle; the heaving carotid arteries in cases of aortic regurgitation; may any of them afford us valuable hints as to the direction of our further inquiries.

§ 33. **Palpation and the localisation of the apex** (see Figs. 9 and 12). The normal apex-beat besides being seen can be felt quite distinctly, first by the flat of the hand, and then by localising it with the finger-tips. In an adult male it is situated about  $1\frac{1}{2}$  inch below and  $\frac{1}{2}$  inch to the inner side of the nipple-line, at a distance of about 3 inches from the mid-sternal line. *These and other cardiac measurements vary with the age,<sup>1</sup> and proportions of the patient*—facts which are apt to be forgotten. The most external portion of the apex-beat should be marked by a dot with an aniline pencil. The localisation of the apex is a most important matter; and there are three principal features to observe about it—its POSITION, its FORCE, and its EXTENT. You may also note in passing whether a *thrill* can be felt with the flat of the hand. It is important to bear in mind that the apex-beat is considerably modified if the apex happen, as it not unfrequently does, to pulsate *precisely behind a rib*. It is only when the apex beats in an intercostal space that these three features can be satisfactorily made out, and this source of fallacy should be remembered and allowed for. It can sometimes be felt more distinctly when the patient is asked to lean forward.

In HYPERTROPHY of the l. ventricle the heart impulse is displaced downwards and outwards, and it is forcible and heaving. In

<sup>1</sup> The position of the heart is considerably modified in childhood. The l. border comes out to the nipple line, and the r. border extends to the r. edge of the sternum; the apex beats almost directly below the nipple, behind the 5th rib, or may be in the 4th interspace.

hypertrophy of the r. ventricle there is pulsation in the epigastrium and in the lower l. interspaces, but the apex is in its normal site. With DILATATION the apex-beat is diffuse and wavy.

The apex is DISPLACED *downwards* in cases of emphysema or pleurisy with effusion; if the latter be on the left side the apex may even be displaced beyond the right border of the sternum (see Fig. 44). The apex is displaced *upwards* in pericardial effusion,

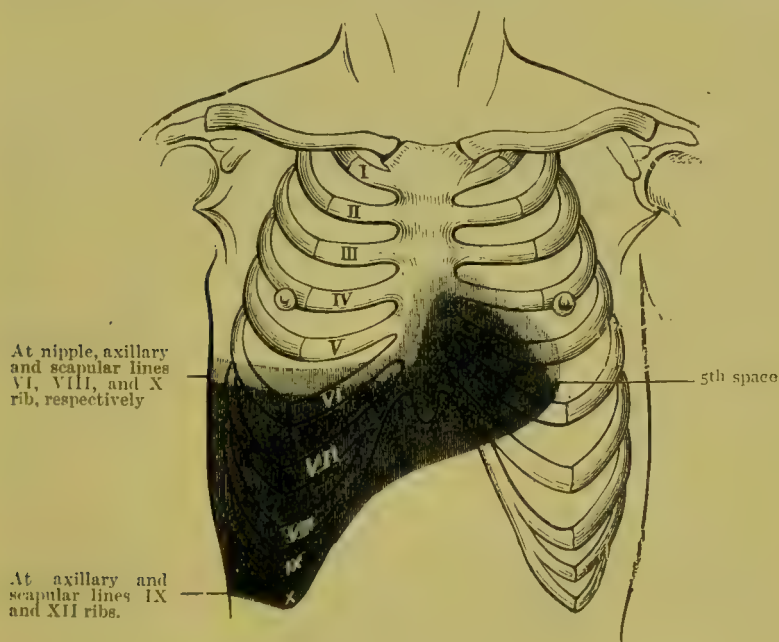


Fig. 9. **Superficial and deep dulness of Heart and Liver.** The superficial area of cardiac dulness is a triangular one, with the apex upwards. The measurements of this area in a person of average size are  $3\frac{1}{2}$  inches transversely, and  $2\frac{1}{2}$  to 3 inches vertically along the left border of the sternum. The r. border begins at the level of the 4th costal cartilage, and corresponds to a vertical line drawn slightly to the left of the middle line of the sternum. The l. limit starts from the same point and runs outwards along the lower border of the 4th c.c. to nearly its junction with the rib, then bending downwards to apex beat. The lower limit is continuous with the Liver dulness.

retracted lung, abdominal tympanites, or with any abdominal tumour pushing up the diaphragm. The apex beat is OBSCURED by very muscular or adipose chest walls, or emphysema. It is FEEBLE with fatty heart; WAVY in pericardial effusion. With pericardial adhesions there is a *systolic retraction* of one or more interspaces.

**THRILLS.** Of endocardial thrills, the presystolic thrill *felt at the apex*, due to mitral stenosis, is the most common. More rarely there is also a thrill in the *pulmonary area* (Fig. 12) due to mitral stenosis. The systolic thrill at the *aortic area*, due to aortic



stenosis, when present, is very marked. A diastolic thrill can be felt over the *manubrium* in advanced cases of aneurysm or aortic regurgitation. Mitral regurgitation is occasionally accompanied by a *systolic thrill at the apex*. A widespread thrill may be present with pericarditis.

§ 34. **Percussion** of the *superficial* area of the præcordial dulness, *i.e.*, area not covered by lung. In mapping out this area the percussion stroke should be *very much lighter*, and more superficial than that applied when examining the lungs, liver, spleen, or other deep-seated solid organs (see Fig. 9). The superficial area which is here referred to is a triangular one with the apex upwards. The measurements of the dull area in a person of average size are  $3\frac{1}{2}$  inches transversely, from the mid-sternal line; and  $2\frac{1}{2}$  to 3 inches vertically, along the left border of the sternum. Its boundaries are given in Fig. 9. The percussion note over the sternum is very different to that elicited over the chest beside it—it is of a much higher pitch—consequently, we cannot compare the percussion note in these two situations. We ought, therefore, to percuss upwards and downwards in a vertical line along the sternum to ascertain if any part of it is duller than normal.

The area of *deep-seated* cardiac dulness is  $\frac{3}{4}$  inch larger on each side and 1 inch larger upwards, than the superficial area. This dulness, due to that part of the heart which is covered by lung, is brought out by much more forcible percussion. Its limits are less certain and therefore less useful for diagnosis than the superficial area.

**Method.** The student should lose no opportunity of PERCUSSING THE NORMAL heart and of attending to the following points:—(i.) *Having first localised the apex-beat*—begin outside the cardiac area in a perfectly resonant area. The l. hand should be placed flat and *firmly* upon the chest-wall parallel to the margin of dulness to be made out, and moved a quarter of an inch at a time, *always parallel to that margin*, towards the centre of the heart. (ii.) Use only one finger—the second of the r. hand—as a hammer, making a short sharp tap with the finger *tip*. The percussing finger should rebound immediately, “*staccato*,” as pianists say. The movement should be made from the *wrist*, or from the knuckle (metacarpo-phalangeal joint) as in playing the piano, and the tap should be a light one. If it be too heavy the deep outline of the heart will be brought out, and that area is larger; and the delicate shades of intensity and pitch will be lost. (iii.) By listening attentively to the sound elicited it will be noticed that it is dull and flat over the heart, like that produced by striking any solid object: but louder and more resonant outside the area, like the sound produced by striking an empty barrel. It is only possible to define in this way the right, the upper, and the left limits of the dull area, because at the lower limit the cardiac dulness is continuous with that of the liver. Mark with a blue

aniline pencil the right or sternal border in two places. The curved upper and left border of the dulness should also be marked by a pencil in two positions, viz., close to the l. side of the sternum, and in another place near the nipple; these can then be joined and continued to the apex-beat.

FALLACIES. It should be remembered that cardiac enlargement may be *obscured* by the hyper-resonance of emphysematous lungs, and under these circumstances, enlargement of the heart or pericardium is very difficult to make out; we have then to rely upon other means than percussion. On the other hand, cardiac enlargement may be *simulated* by a fibrous retraction of the left lung, the heart nevertheless remaining of normal size; or, thirdly, the heart may be *displaced* by an aneurysm or other mediastinal tumour pushing forward, and making the præcordial area appear larger. One or other border of the area of dulness may be *obscured* by pleuritic effusion; and a method of defining the l. border of the heart by means of a tuning fork and ausculto-percussion is described in § 89 (p. 181). Ascites or pleural effusion actually *displace* the heart, as in the case given in Fig. 44, p. 181.

The boundaries of the præcordial dulness are of great importance—so much so that we are enabled, as we shall see, to classify both acute and chronic diseases of the heart by the presence or absence of enlargement.

THE CHIEF CAUSES OF ENLARGEMENT of the præcordial area of dulness are two in number—EFFUSION into the pericardium and ENLARGEMENT of the heart. The latter may be due to *hypertrophy* or to *dilatation*, or, more commonly, to a combination of the two. This enlargement may involve any one, or more than one, of the cavities of the heart.

§ 35. **Resistance to Palpation.** Thus far reference has only been made to the sound which is heard by *percussing* the chest, dull or resonant as the case may be. But it has often been noticed that physicians who are losing their hearing can nevertheless map out the boundaries of solid organs. This they do by appreciating the greater "resistance" to the stroke of the finger which is offered by a solid than by a hollow substance. After some practice the student will learn to appreciate this quality of resistance, but it is difficult at first. It may be elicited in the process of percussion with two hands, as above described; or else, and perhaps this is better, by *lightly pressing the bare chest with a finger tip of one hand*. Let the student place the first and second finger tips of the right hand in the third and fourth intercostal spaces to the l. of the sternum, and lightly press alternately with each finger—he will find a difference of resistance. If he presses lightly with one finger tip in various other positions, of course avoiding the cartilages, ribs, and other bony prominences, he will find the resistance varies considerably in different places. The faculty of appreciating this quality of resistance varies greatly in different individuals, but in some it is so refined that they can thus delineate the boundaries of solid organs, with the greatest precision, without the necessity for percussion. This subject is admirably discussed by Dr. Robert Maguire in the "Practitioner" of April, 1897, p. 371.

§ 36. For **Auscultation**, much practice is required, and once more I must warn the student never to miss an opportunity of

listening to the sounds of the heart, particularly the *normal* sounds. The whole cardiac cycle—*i.e.*, contraction (systole) and dilatation (diastole)—occupies about one second (Fig. 10). The first sound is due to the contraction of the ventricular muscle or to the

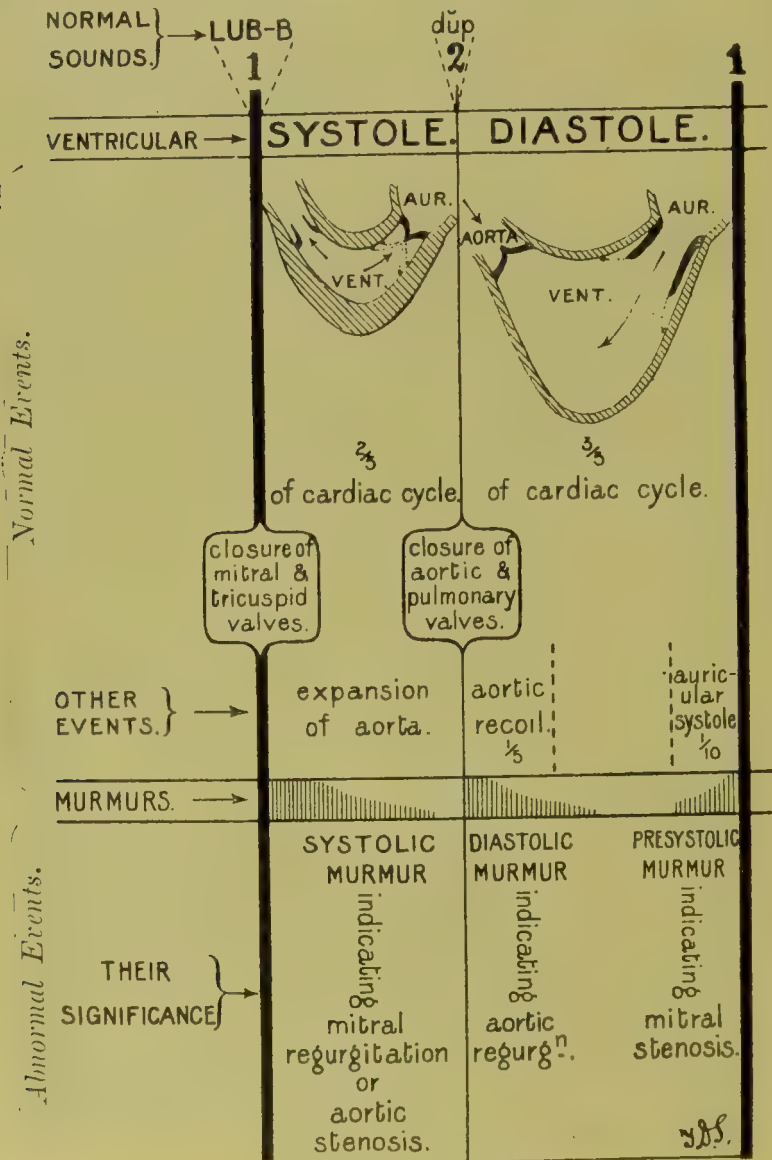


Fig. 10. Diagram of a Cardiac Cycle, showing various events, and their duration; how the different murmurs are produced; and their clinical significance. The student should study this and Fig. 12 very closely.

closure of the mitral and tricuspid valves (or to both), and occupies about  $\frac{1}{10}$  second; then comes a very brief interval, say  $\frac{1}{10}$  second; followed by the second sound ( $\frac{2}{10}$  second), which is due to the closure of the aortic and pulmonary valves; and finally the diastolic interval,  $\frac{3}{10}$  second (Fig. 10 should be carefully studied by the student).

**Methods.** If no stethoscope is handy, cover the patient's skin with a soft handkerchief, preferably silk, and apply the ear; but a stethoscope localises the sounds better. Personally, I prefer the old-fashioned wooden stethoscope if the ear-piece fits my ear well. But the binaural stethoscope is very useful for examining infants, or whenever there is any noise in the room or in the street, though it always has the disadvantage of giving more resonance-tones (echoes arising in the cup and tubes). Place the small end of the stethoscope on the chest over the apex-beat, so that *the rim touches all round*, and then adjust *your ear to the stethoscope*, not the stethoscope to your ear. The common fault is to allow one side of the chest-end to be raised off the chest in the process of adjusting the stethoscope to the ear. Do not allow the weight of your head to rest on the stethoscope: it pains the patient, and you do not hear as well. It is a good plan to keep your fingers on the pulse (wrist or carotid) whilst auscultating.

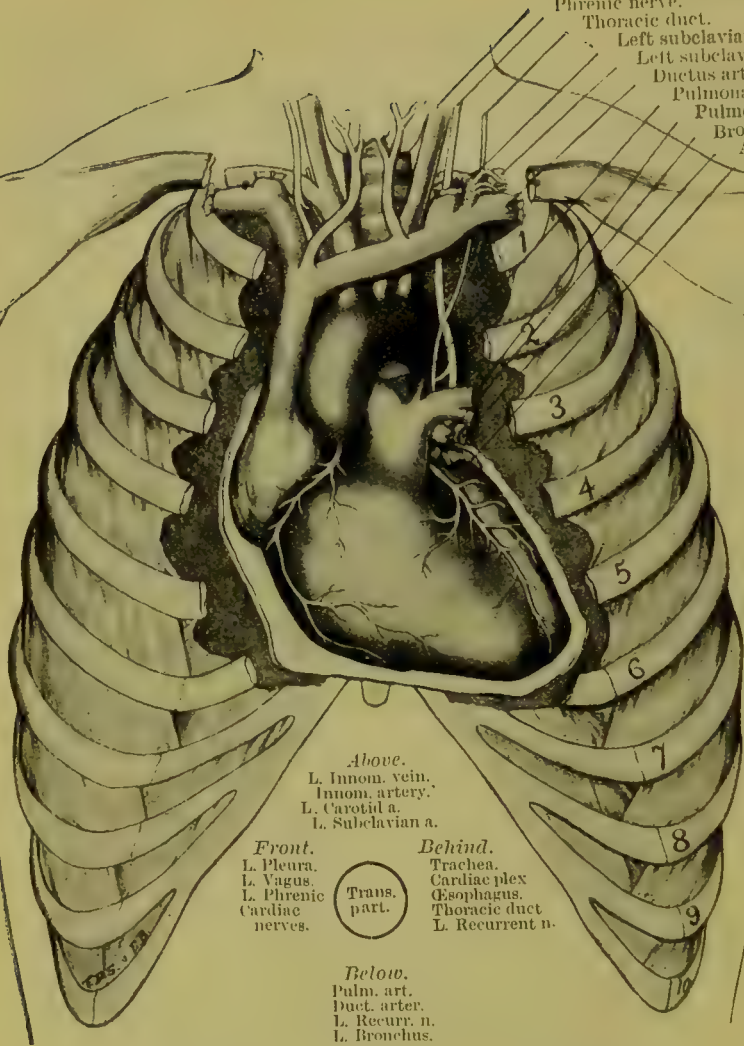
Listen, *first*, to the sounds at the *apex* and all round its neighbourhood. Notice that the first sound is normally longer and duller than the second,<sup>1</sup> and that the two sounds somewhat resemble lubb ( $\frac{1}{10}$  second), dup ( $\frac{2}{10}$  second). Then, *secondly*, listen at the base; place the instrument over the second r. intercostal space, close to the sternum, near the position of the aortic valves; listen at that spot, and in other places around, as indicated in Fig. 12. The student will notice that the actual position of a valve, Fig. 11, does not correspond precisely with the position of the maximum intensity of the sounds produced at that orifice (Fig. 12). This should always be the order of investigation, and by listening in this manner, we are enabled to make two important observations: (i.) the presence or absence of a MURMUR (peri- or endo-cardial); (ii.) whether either sound is unduly shortened, prolonged, emphasized, or reduplicated.

If a murmur or *bruit* be discovered, there are four qualities to be observed concerning it: (i.) its rhythm (*i.e.*, whether it replaces the 1st or 2nd sound); (ii.) its position of maximum intensity; (iii.) the direction in which it seems to be propagated (*i.e.*, continues to be audible); and (iv.) its quality as regards roughness. These may be briefly summarised as Rhythm, Position,

<sup>1</sup> The first and second sounds correspond to G and B-flat respectively, below the middle octave.



Internal carotid artery.  
Vagus nerve and recurrent laryngeal branch.  
Phrenic nerve.  
Thoracic duct.  
Left subclavian artery.  
Left subclavian vein.  
Ductus arteriosus.  
Pulmonary artery.  
Pulmonary vein.  
Bronchus.  
Auricular appendix.



Above.  
L. Innom. vein.  
Innom. artery.  
L. Carotid a.  
L. Subclavian a.

Front.  
L. Pleura.  
L. Vagus.  
L. Phrenic  
Cardiac  
nerves.

Trans.  
part.

Behind.  
Trachea.  
Cardiac plex  
Oesophagus.  
Thoracic duct  
L. Recurrent n.

Below.  
Pulm. art.  
Duct. arter.  
L. Recurr. n.  
L. Bronchus.

Front.  
Pulm. art.  
R. Auric. app.  
Thymus Gld. and  
Pericard.

Right.  
Sup. Vena  
R. Auricle.

Ascend.  
part.

Left.  
Pulm. art.

Back.  
R. Pulm. vessels.  
Root of R. lung.

Front.  
Pleura.  
Root of L. lung.

Right.  
Oesophagus.  
Thoracic D.

Descend.  
part.

Left.  
Pleura.

Back.  
L. side body of 4th Dors. Vert.

Fig. 11. The Heart and great vessels in situ with lungs turned back stretched from the Cadaver. R. vent. forms greater part of the ant. surface of the heart; above and to r. of this, is the r. auricle into which the sup. vena cava collects the blood from the subclavian, inferior thyroid and jugular veins. Passing out from and above the r. ventr. is the pulmonary artery, above which again is the remains of the ductus arteriosus, connecting it with the arch of the aorta. Just to the left of the pulmonary artery, the left auricular appendix peeps round the corner. The arch of the aorta is seen, coming forward from the l. vent. (which is at back, and therefore only seen at l. margin of heart); and from its upper convexity arise in order, the r. coronary artery, innominate artery, left carotid, and l. subclavian. The trachea is seen behind the vessels, and the phrenic and vagi nerves are seen at the sides, those on the left passing down in front of the aorta behind the root of the left lung. The relations of the Ascending, Descending, and Transverse portions of the aorta are given diagrammatically below.



Propagation, and Character. A systematic method of this kind is easily acquired, and the habit will afterwards be of the greatest

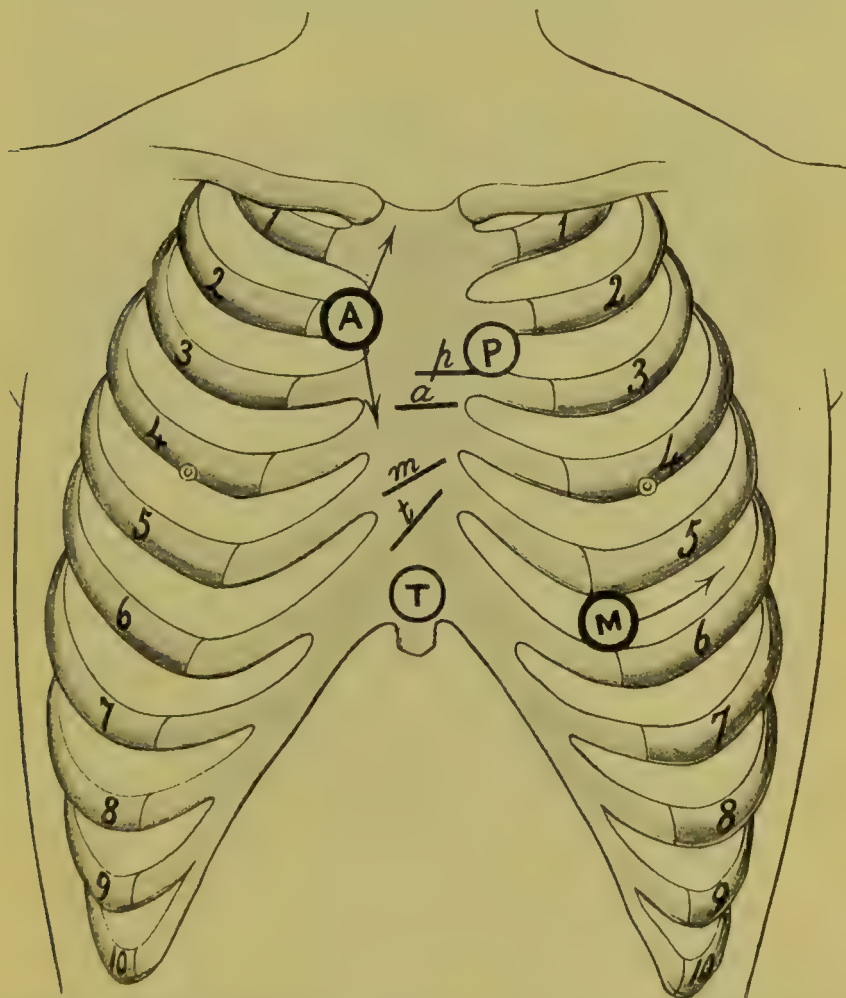


Fig. 12. DIAGRAM SHOWING THE SITUATION OF THE Cardiac valves AND THE position IN WHICH THE SEVERAL murmurs ARE HEARD LOUDEST.

*p* = pulmonary orifice, at level of upper border of 3rd l. costal cartilage.

*a* = aortic orifice at level of lower border of 3rd l. costal cartilage.

*m* = mitral orifice at level of lower border of 4th l. costal cartilage.

*t* = tricuspid orifice at level of 4th interspace, lying obliquely behind the sternum.

The positions where the sounds produced at the various orifices are best heard are indicated by the letters enclosed in circles; the arrows mark the direction in which murmurs produced at the corresponding orifices are conducted.

**M**, Mitral murmurs are best heard at the mitral area, *i.e.*, the apex.

**A**, Aortic " " " " aortic " " 2nd r. c.c.

**P**, Pulmonary " " " " pulmonary area, *i.e.*, 2nd l. intercostal space.

**T**, Tricuspid " " " " tricuspid " " at lower end of sternum.

use. It is by the cultivation of such habits that the clinical observer becomes evolved.

## ALTERATIONS OF THE HEART SOUNDS AND THEIR SIGNIFICANCE.

1. *Murmurs* heard with the *first* sound are *systolic* in rhythm ; murmurs with the *second* sound are *diastolic* (see Fig. 10). There are 4 fundamental facts which the student must never forget, viz.—  
*Systolic* m. at apex continued into axilla ..... = *mitral regurgitation* ;  
*Presystolic* m. limited to apex ..... = *mitral stenosis* ;  
*Systolic* m. in aortic area, conducted along carotids = *aortic stenosis* ;  
*Diastolic* m. at aortic area, conducted down sternum = *aortic regurgitation*.

2. *Accentuated second* sound in the *aortic* area, at the base of the heart is met with when the arterial tension is high (§ 64), or in aortic aneurysm. In the *pulmonary* area an accentuated second is due to increased blood pressure in the lungs, as in mitral valve disease.

3. A *reduplicated second* sound at the *base* of the heart is found when the aortic and pulmonary valves do not close synchronously, as when the pressure in either the arterial or the pulmonary system is unduly high (as in 2). When heard a little to the r. of the *apex* it is very characteristic of mitral stenosis. A *reduplicated first* at the apex is sometimes found with high arterial tension.

4. *Short, clear, sharp sounds* are found with cardiac dilatation, while *feeble* sounds occur with fatty heart. Emphysema, excess of adipose tissue, or pericardial effusion obscure the sounds and give the impression of feebleness.

5. A *prolongation* of the first or second heart sound is sometimes spoken of, but it is difficult to know where to draw the line between a prolongation and a murmur. A “booming” first is found with hypertrophy.

**FALLACIES.** Under ordinary circumstances the respiratory do not interfere with the cardiac sounds, but if they do, the patient should be asked to stop breathing for a few seconds. It is often wise to do this in any case, because sounds originating in the bronchi or pleura may be mistaken for cardiac murmurs, but *they cease, of course, when the patient stops breathing*. On the other hand, if a murmur be very feeble and doubtful, the heart sounds may be exaggerated by causing the patient to take some exertion, such as running upstairs.

§ 37. **The pulse** affords one of the best possible indications as to how the heart is performing its work, as to what remedies, especially alcohol, are indicated, and finally as to how the therapeutic measures we have already adopted are answering their purpose. The pulse also gives an important clue as to the nature of the cardiac lesion ; thus, in mitral regurgitation the pulse is

1 Stenosis (*στένωσις*, to contract) indicates obstruction or narrowing of an orifice ; regurgitation indicates a backward flow from imperfect closure of the valves. The small type is here used, not to indicate secondary importance, but as a matter of convenience.

usually rapid, irregular and of low tension, while in mitral stenosis it is regular but small, thready, and incompressible; in aortic regurgitation it presents a collapsing ("water hammer") character, while in aortic stenosis it presents exactly the opposite feature,

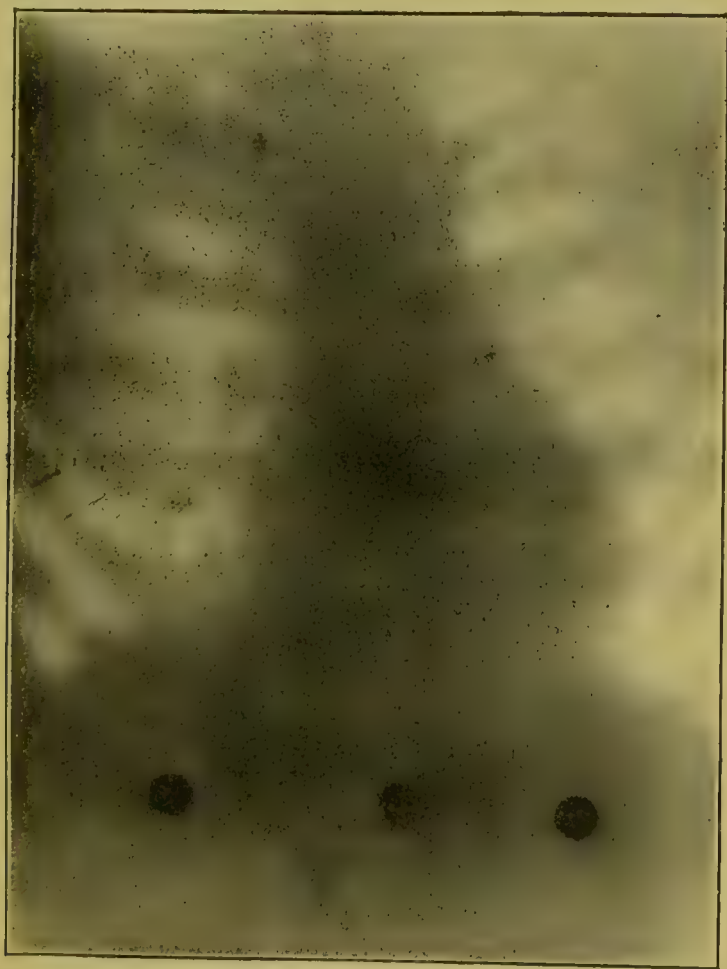


Fig. 13. **Skiagram of the heart, showing dilatation of the r. ventricle.** It was taken with the patient's clothes on, and the buttons are shown. Made by Dr. John Macintyre, of Glasgow, who kindly lent it to the author.

being sustained. The pulse will be dealt with fully in a separate chapter (Chap. V.), but there are three important hints which may be mentioned here—viz., (1) Do not examine the pulse until the nervousness at first excited by your visit has passed off. (2) Generally keep your fingers on the pulse while auscultating

the heart. (3) In all cardiac cases the rate, rhythm, force, and tension of the pulse should be frequently noted.

§ 38. **Ausculto-percussion.**—In doubtful cases where it is desired to delineate the outline of the heart with greater precision (for instance to observe the immediate effects of the Nauheim baths on a dilated heart) this method is now again coming into use, but it requires considerable practice for its efficient application. A binaural stethoscope is desirable. The chest-piece of the stethoscope may be placed just above the right nipple, and then a tap made with a light hammer, or the end of an aniline pencil shod with an india-rubber or metal cap, over the centre of the cardiac area; then move the stethoscope gradually up towards the heart, tapping continuously with the hammer. As the stethoscope crosses the *deep* boundary of the heart, a distinct alteration of the sound conducted to the ear will be observable by one well practised in the procedure, and its position can be marked with the other end of the pencil. Then place the stethoscope close to the l. of the manubrium and work downwards in the same way, and so on, all along the r. and l. boundaries. Guineá de Mussý, who first introduced this method, employed an assistant to tap on the back, while he shifted the stethoscope from place to place on the front of the chest. He employed this same method for examining the lungs.

§ 39. **Radiography.**—In 1896 Professor Röntgen discovered that the X rays of the light spectrum, though invisible to the eye, are capable of affecting sensitised photographic plates; and that they also are able to traverse, in greater or less degree, all the fluid and solid tissues of the body. But the bones, and any metallic substances, such as a bullet, are relatively opaque to these rays, and come out as shadows in the photograph (see Fig. 13). It was subsequently discovered that the outline of some of the more solid viscera could also be revealed by adopting certain precautions in arranging the apparatus.<sup>1</sup>

**Aspiration of the pericardium** is a method sometimes used for ascertaining what is the nature of the fluid in cases of Pericardial Effusion: see § 41, p. 70.

### C. DISEASES OF THE HEART AND PERICARDIUM; THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT.

§ 40. **Classification.** For practical purposes, diseases of the heart and pericardium are best grouped into ACUTE and CHRONIC; and each of these may be subdivided into those attended by *enlargement of the precordial dulness*, and those *not necessarily so attended*.

AREA OF DULNESS INCREASED.

AREA OF DULNESS NOT  
NECESSARILY INCREASED.

#### Acute.

I. Acute pericarditis with  
effusion.

I. Acute endocarditis.  
II. Neuro-palpitation (Paroxysmal  
Tachycardia).  
III. Angina Pectoris.

#### Chronic.

I. Cardiac Hypertrophy.  
II. Cardiac dilatation.  
III. Hydropericardium.  
IV. Congenital Heart disease.  
V. Aortic aneurysm or other  
mediastinal tumours (§ 56).

I. Cardiac valvular disease.  
II. Fatty Heart.

This is not, of course, a disease of the heart proper; but it is included here because its existence is often revealed

by finding enlargement (and perhaps *distortion*) of the precordial dulness.

<sup>1</sup> Dr. John Macintyre (Glasgow)—*The Lancet*, 1896, ii. 567; Dr. Hugh Walsham, *The Lancet*, Nov. 3, 1900; and see Appendix at end of this book.

§ 40a. **Routine Procedure.** FIRST: WHAT IS THE PATIENT'S "LEADING SYMPTOM"? It may be that the patient voluntarily complains of one of the symptoms discussed in Section A, and in this way has directed our attention to his heart. If not, we must ascertain, without putting "leading questions," what is his "chief symptom." Breathlessness is the most constant symptom in cardiac disease, and in more advanced stages we meet with dropsy and cyanosis.

SECONDLY: Having obtained this clue we follow it up by asking a few details, in chronological order, of the HISTORY OF THE ILLNESS. In this way we *ascertain whether the disease be acute or chronic*—a most important matter, because for clinical purposes diseases of the heart and pericardium may be primarily divided into acute and chronic. The Previous and Family Histories may also be inquired into. In all cardiac cases it is important to know whether the patient has ever had acute rheumatism, this being the most frequent cause of cardiac valvular disease. Ascertain also if there be any heart disease in the family.

THIRDLY: THE EXAMINATION OF THE HEART, and especially the decision as to *whether there is any enlargement or not*. The routine method consists of the following procedures:—

1. An examination of the apex-beat (by *inspection* and *palpation*);
2. The mapping out of the area of præcordial dulness (by *percussion*) (see Fig. 9).
3. Listening to the heart sounds (*auscultation*); and
4. The examination of the *pulse*.

The chest should always be stripped and a thorough examination made. An attempt to examine a female patient should never be made without the removal of the corsets.

If the symptoms of which the patient complains point to some **chronic cardiac disease**—*i.e.*, they have come on gradually and are unattended by pyrexia or other constitutional disturbance—the reader should turn to the **chronic diseases** (§ 45).

If, on the other hand, the disease is of an **acute** character—*i.e.*, it has come on recently or suddenly, and is perhaps attended by pyrexia and other constitutional symptoms—it is one of four diseases: I. ACUTE PERICARDITIS; II. ACUTE ENDOCARDITIS; III. PAROXYSMAL TACHYCARDIA; or IV. ANGINA PECTORIS.



I. If the præcordial area of **dulness** be increased, the shape of the dulness being PYRAMIDAL, with the point upwards, and if the temperature be elevated, the disease is almost certain to be ACUTE PERICARDITIS. The only possible alternative is that the case may be one of the chronic diseases attended with enlargement (§ 46), upon which an acute condition has supervened.

§ 41. **Acute Pericarditis** is an acute inflammation of the pericardial sac. The disease has two stages; the *first* (which rarely lasts longer than 12 to 48 hours) precedes, and the *second* follows, the effusion of fluid into the pericardial sac. It is not frequently met with as a primary affection. It supervenes during the course of many different diseases, and the symptoms of these may mask its onset. Certainly its most common cause is rheumatic fever, and it should be remembered that it may be the first manifestation of this affection. We should always examine the heart daily in rheumatic fever, and in acute renal affections because, in these, acute pericarditis may come on insidiously, without pain or tenderness, its advent being marked perhaps only by the occurrence of delirium, so rare otherwise in acute rheumatism.

*Symptoms.* 1. The patient wears an anxious, troubled look, and the cheeks are flushed; there are fever and a rapid pulse; the breathing is rapid, and he complains of severe pain over the heart, increased by pressure, movement, or respiration. 2. *Physical Signs.* The præcordial dulness is not increased in the first stage—*i.e.*, during the first day or two—but a loud harsh *double murmur*, “to and fro,” like a saw, is heard on auscultation. This may be distinguished from a murmur produced *within* the heart by (i.) always being double (*i.e.*, accompanying both sounds of the heart); (ii.) the roughness of the first is continuous with that of the second sound, without any diastolic pause; (iii.) it is always loudest at the root of the great vessels, over the third l. costal cartilage; (iv.) it varies in its character from time to time, and is increased by gentle pressure; (v.) pressure will also elicit another differential character—*viz.*, that the disease is usually accompanied by tenderness, as well as pain. The differentiation between Peri- and Endo-cardial murmurs is so important that it is also given in a tabular form below (p. 71). To distinguish pericardial from

pleuritic friction is very easy, because the latter ceases if the patient holds his breath. Note that as the effusion occurs the murmur becomes less distinct, but it is again intensified as the effusion clears up.

3. *Second stage*, or stage of pericardial effusion. The inflammation may subside; but, more frequently, in the course of a day or two, effusion of fluid occurs, and the pain and tenderness diminish. The murmur becomes less audible, though it can still be heard at the root of the heart. The temperature may fall a

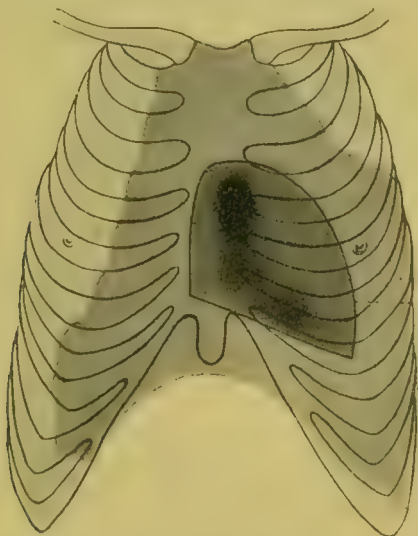


Fig. 14. Diagram from a case of Rheumatic Pericarditis with effusion. Eliza P., æt. 27. The dark area indicates precordial dullness. The larger area of lighter shading gives the area over which pericardial friction could be heard (the loudness is indicated by the depths of the shading). It is usually taught that one of the features distinguishing peri- from endo-cardial murmurs is the limitation of the former to the precordial region; but I have many times satisfied myself that this is not so, and this case is one of several examples I have met with, verified by autopsy. This case was under the care of Dr. W. M. Ord, when I was his house-physician.

little, but the breathlessness and other symptoms continue. *The increased area of dullness*, due to pericardial effusion, may be greater than the enlargement from any other cause, and it has moreover three very well-defined characters. (i.) It is of *triangular shape*, with apex upwards, reaching to the third or even second costal cartilage. (ii.) As the root of the heart is fixed to the pericardium, when the sac fills the whole heart becomes raised, and, therefore, *the apex beats above and to the l. of its normal position*. (iii.) *The dullness extends to the left of the apex-beat*. The heart

sounds may be diminished at this time, because they are transmitted through fluid. It is possible by the height of the dulness along the sternum, which should be watched each day, to determine the amount of fluid present.

*Etiology.* Pericarditis may attack any age and either sex, but is almost always preceded and caused by some other disease. It is doubtful if idiopathic pericarditis ever occurs. The causes of pericarditis may be ranged under four heads: 1. *Injury*. 2. *Certain morbid states of the blood*, which in order of frequency in this country are: Acute Rheumatism, uræmia, pyæmia, various acute infectious fevers—*e.g.*, scarlatina, variola, typhus, typhoid, and epidemic influenza (Barlow), and some constitutional diseases—*e.g.*, scurvy and gout. 3. *Morbid growths*—*e.g.*, tubercle and cancer, in both of which the process tends to be subacute, and is accompanied by a large amount of fluid. 4. *Extension* from adjacent organs, amongst which may be mentioned—pleurisy or pleuro-pneumonia especially on the left side; intra-thoracic aneurysm (pericarditis may be the precursor of rupture into the pericardium); solid intrathoracic tumours; perforating ulcer of the œsophagus; various diseases below the diaphragm—*e.g.*, hydatid or abscess of liver.

*Course and prognosis.* The duration of acute pericarditis with effusion varies widely, but it averages about 15 to 25 days. It may undergo resolution with or without the formation of adhesions (Adherent Pericardium *Ic.* below); or result in chronic effusion (Hydropericardium, § 48); or become purulent (Pyo-pericardium *Ia.* below). Pericarditis with effusion is always a serious malady, but the prognosis depends much on the disease which it complicates, the general condition of the patient, and the evidences of cardiac embarrassment—namely, feebleness, rapidity, and irregularity of the pulse. Pericarditis complicating rheumatism, like the other complications of that disease, tends to recover, but it may leave a weakened heart, and lead to cardiac dilatation. In renal disease it is a serious though often latent affection; and in pyæmia, when it is generally purulent, it adds to the gravity of that serious disorder. In infancy and in debilitated patients it is also grave.

*Treatment.* In the inflammatory stage the patient should be

kept in bed absolutely *without movement*, on light fluid diet; and cotton-wool, a poultice, or warm fomentation applied to the præcordium. This usually gives more relief than the ice-bag which is recommended by some (*e.g.*, Dr. Lees), though this undoubtedly relieves the symptoms. If the distress is great, wonderful relief is obtained from four or five leeches over the præcordium. If cyanosis, orthopnœa, and irregular pulse are present, indicating considerable cardiac embarrassment, bleeding (four to six oz.) is a prompt and efficacious measure. Opium (gr.  $\frac{1}{4}$  quartis horis), or morphia hypodermically, is of great value for the pain and distress. Small doses of chloral may be given for the restlessness if the circulation is well maintained. Effervescing salines should be administered. Digitalis is given in small frequent doses for cardiac failure, and stimulants, ether and strychnine, according to the state of the pulse. Digitalis must be very carefully watched, lest it increase the cardiac embarrassment. For hyperpyrexia and delirium some recommend the graduated bath, but the necessary movement is a grave objection; and we have remedies quite as valuable in antifebrin, phenacetin, aconite, quinine, and opium.

*Treatment for the cause* of the pericarditis may be combined with the foregoing—*e.g.*, sodium salicylate (with caution, lest it depress the heart) or full doses of alkalies for acute rheumatism—the latter being better because salicylates have but little controlling action over the effusion: diuretics and hot-air baths for renal disease: quinine in large doses for pyæmia. In the stage of effusion, free blistering promotes absorption; but it must be remembered that renal disease is a contra-indication to blistering. If the effusion becomes chronic, pot. iod. (gr. v., t. d.) and diuretics may be given (F. 55). Iodine paint and other local counter-irritants are also useful.

PARACENTESIS PERICARDII. If, at any time, the effusion be considerable, and the cardiac embarrassment very great, as evidenced by severe dyspnœa, and a rapid, irregular, low-tension pulse; if leeches and bleeding have failed to give relief, exploration with a hypodermic syringe, under strictly antiseptic precautions, may be practised *to ascertain the nature of the fluid*.<sup>1</sup> If pus be found, paracentesis should certainly be performed, and the

<sup>1</sup> The operation of paracentesis has now been performed sufficiently often to make it trustworthy. See cases reported by H. B. Robinson, *Brit. Med. Journ.*, 1898, vol. ii., p. 1605; and Foxwell, *B. M. J.*, 1897, vol. i., p. 1350; and footnote, p. 70.

operation may be done without fear, if rigid antiseptic precautions be employed, and the point of the trochar kept away from the viscus. Incise the integument in the fifth l. interspace, close to the sixth rib, 2 to 2½ inches to the left of the middle of the sternum (in an adult of average size). Insert the trochar and cannula perpendicularly to the surface; withdraw the former directly it pierces the wall. Eight or twelve, or even forty ozs. (in a chronic case) may be thus gradually removed.

**Ia. Pyo-pericarditis.** Sometimes in debilitated children, and in the course of scarlatina, always in the pericarditis of pyæmia, and under some other conditions, the fluid in the pericardium takes on a purulent or sero-purulent character. This condition is usually revealed (as in a collection of pus in other parts of the body) by the occurrence of (1) shivering attacks, (2) profuse perspirations, and (3) a temperature with wide variations in the course of a few hours, in addition to the clinical features of acute pericarditis above described. But it is very difficult to diagnose because the *friction sound is usually absent*. If contracted it is usually fatal.

Pyo-pericarditis is the form which pericarditis frequently assumes in infancy, and is then extremely difficult to diagnose. In addition to the small measurements with which we have to deal, the l. lung may become adherent to the chest-wall early in the disease, and so prevent the enlargement of the præcordial dullness being manifest. It is only by the profound disturbance of the circulation, progressive weakness, and anæmia, combined with sweatings, that we can assume the presence of pus.

*Treatment.* Quinine in large doses, phenacetin and like remedies may be administered, but a large hypodermic syringe, rendered thoroughly antiseptic, should be very carefully introduced, whenever the existence of pyo-pericardium is suspected. If the fluid withdrawn be of a purulent nature, paracentesis, or better still free drainage, should be effected.<sup>1</sup>

**Ib. Latent Pericarditis**—*i.e.*, pericarditis without *symptoms* (though not necessarily without physical signs). In most patients in whom we find a pericardial effusion, a history of acute pericarditis is obtainable; but it is a fact not sufficiently recognised that pericarditis may have come on quite insidiously, without any acute symptoms. The effusion may be discovered when examining the heart as a matter of routine, or perhaps it may be for the first time, at the autopsy. Nevertheless, in such cases the presence of flakes of fibrin, and the dull thickened serous surfaces, reveal its inflammatory origin. Moreover, I have, in the p.m. room, on more than one occasion, found a totally adherent pericardium in a patient in whom the most careful inquiry had failed to reveal any symptoms pointing to the heart during life. It is a latent pericarditis of this kind which ordinarily complicates RENAL DISEASE and UREMIA. In ACUTE RHEUMATISM also *its advent may be indicated only by delirium or vomiting*; and GOUTY persons also may be attacked by this latent disease after exposure to chill.

Pericarditis occasionally results in two somewhat serious and happily rare conditions. Adherent Pericardium and Mediastina Fibrosa.

**Ic. Adherent Pericardium** is usually *unattended with any enlargement of the præcordial area of dullness*, and may be readily overlooked during life. It depends a good deal on the nature of the adhesions, whether it gives rise to symptoms or not. Thin areolar connections of limited extent are of no consequence, but dense, fibrous, universal

<sup>1</sup> See Discussion at Clin. Soc., *The Lancet*, Dec. 22, 1900.



adhesions embarrass the heart, and may give rise to palpitation, irregularity of action, and syncope; and atrophy of the organ may result.<sup>1</sup> The only signs by which it may be suspected during life are: 1. A recession, instead of a protrusion, at the cardiac apex with each systole, and perhaps a synchronous depression of one or two of the inter-spaces above. 2. No alteration of the position of the apex beat during deep inspiration. 3. There is sometimes a history of the attack of pericarditis which has caused the adhesions. "*Milk-Spot*" is a thickening of the visceral pericardium on the anterior surface of the heart. It will be referred to under "systolic murmur," which is the chief symptom to which it gives rise (§ 50).

Id. *Mediastina Fibrosa* is a term which may be applied to the extremely rare condition first described by Greisinger, in which the fibrous adhesions of the pericardium extend to and involve the anterior mediastinum. Its existence may be suspected by an irregular intermittent pulse, a stoppage of the pulse during each inspiration (*pulsus paradoxus*), and distension of the jugular veins.

We now pass from the only acute disorder necessarily attended by increase in the area of præcordial dulness, to those **acute disorders** in which the **dulness is not necessarily increased**—viz., II. ACUTE ENDOCARDITIS, III. NEURO-PALPITATION, and IV. ANGINA PECTORIS.

II. *The patient is in evident distress; his TEMPERATURE IS ELEVATED, and on auscultating the chest there is a ROUGHNESS added to the heart sounds,—the disease is ACUTE ENDOCARDITIS. There is just a possibility that it might be Acute Pericarditis with very little effusion. It is not always easy to distinguish endocardial from pericardial murmurs (see Table below).*

TABLE III.—DIAGNOSIS ENDOCARDIAL from Pericardial Murmurs.

ENDOCARDIAL MURMURS.	PERICARDIAL MURMURS.
1. May accompany 1st or 2nd sound only, or both. If double, there is always a short interval of silence between the two bruits.	Always double, and can be heard throughout the diastole as well as the systole, without any interval between the two bruits.
2. Loudest near the valve of origin.	Always loudest over 3rd l. costal cartilage (root of big vessels).
3. May be propagated up into the axilla, or along the aorta and carotids.	Mostly confined to the præcordium. <sup>2</sup>
4. Usually no pain or tenderness.	Usually accompanied by pain.

§ 42. **Acute Endocarditis**<sup>3</sup> is acute inflammation of the lining membrane of the heart. It is unattended by enlargement

<sup>1</sup> Not hypertrophy, as was formerly taught (*e.g.*, by Walshe, "Diseases of the Heart," 1873).  
<sup>2</sup> For an exception to this see Fig. 14, p. 67.

<sup>3</sup> Acute Endocarditis consists of a redness and roughness of the endocardium, particularly over the valves, which are constantly rubbing against each other. In that position wart-like projections or "vegetations" appear when the disease has existed some little time. These changes are most abundant on the lines where the valves touch each other in closing. These vegetations may to some extent subside, but once formed they never entirely

of the præcordial dulness unless some cause for the increase other than the endocarditis exists, such as cardiac hypertrophy or dilatation,<sup>1</sup> or myocarditis. In a very large proportion of cases it complicates some other disease; and, like pericarditis, it is very frequently associated with acute rheumatism; it may even be the first evidence of that disease.

There are two varieties of endocarditis, commonly known as SIMPLE and MALIGNANT, and there are three groups of symptoms found with each.

In SIMPLE or BENIGN ENDOCARDITIS, as in the other variety, (1) the characteristic feature is the *development of a murmur*, usually heard loudest at the apex because the mitral valve is the one most frequently involved in acute rheumatism; but it may be heard in any situation, depending on the valve affected (see Fig. 12), and it may be single or double in rhythm. The murmur has to be diagnosed from that of pericarditis (see Table above), and, if possible, from that due to old valvular disease. In the acute disease the murmur is usually softer and heard over a more limited area; in old valvular disease it is harsher, and is propagated in different directions (*vide* Cardiac Valvular Disease, § 50). The previous history, and the presence of cardiac dilatation, may also aid us considerably.

2. The *Constitutional Symptoms* may be so few and slight, that at the time they may pass almost unnoticed. But since simple endocarditis usually complicates some other disease (*e.g.*, acute rheumatism), the constitutional symptoms largely depend upon the severity or mildness of the primary disease. The onset of the endocarditis in these circumstances may be suspected when there is a sudden increase in the rapidity of the heart, and an additional rise of temperature without apparent cause. Palpitation may be present; but pain and distress about the præcordium are generally absent—a feature worth bearing in mind. In the rare instances in which acute endocarditis occurs primarily, the

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disappear. The resulting chronic thickening, roughness, and puckering constitute chronic endocarditis. Many valuable researches have been made into the pathology of acute endocarditis of late years. It was formerly regarded as a purely *local* disorder, but it is now known to be due to a blood change having specially injurious effects on the endocardium. In the Simple form these effects are limited to this structure, but in the Malignant or Infective type, important generalised effects of a septic character ensue (see footnote, p. 74).

<sup>1</sup> In children acute endocarditis is frequently attended by dilatation, probably due to concurrent myocarditis.

temperature is irregularly intermittent ( $100^{\circ}$ — $102^{\circ}$  F.). The presence of such a pyrexia, and the absence of physical signs, excepting those referable to the heart, are the only data upon which we can rely for the *diagnosis* of the disease.

3. *Emboli* do not usually occur, at any rate until very late, in *simple* endocarditis attacking a heart previously healthy. But when it attacks a heart the seat of old valvular mischief—known sometimes as RECURRENT ENDOCARDITIS—the temperature may vary from  $100^{\circ}$  to  $102^{\circ}$  F. for days, weeks, or even months; and emboli may arise in various situations from the separation of the inflammatory material on the valves. Rigors, with tenderness and enlargement of the spleen, may indicate embolism of that organ; sudden hemiplegia or other nerve troubles may point to embolism in the brain; sudden occurrence of bloody albuminous urine, with a rigor, point to embolism in the kidney; sudden blindness, to embolism of the central artery of the retina; and sudden pain and tenderness in a leg or arm may indicate plugging of one of the arteries, in which case the pulsation will be absent below the blockage.

*Causes of Benign Endocarditis.* A history, or evidence at the time, of the causes of endocarditis may help us in the diagnosis. (i.) The most common of these undoubtedly is rheumatic fever old or recent, and it should be remembered that acute endocarditis may arise quite early in the course of the disease, before the joint lesions are manifest. Exposure to cold is mentioned as a cause, but the endocarditis in such cases is probably of a rheumatic kind. (ii.) Chorea, scarlatina, typhoid, and many other bacterial infections may give rise to endocarditis. (iii.) It is also an occasional complication of syphilitic,<sup>1</sup> cancerous, and other cachectic conditions, chronic alcoholism, and renal disease. (iv.) Valves deformed by acute or chronic endocarditis are predisposed to acute inflammation, and the Recurrent Endocarditis above referred to thus arises. (v.) The patient is generally young, rarely older

<sup>1</sup> H. L., a lad, *æt.* 15, was admitted into the Croydon Hospital in 1882 with intense chlorosis, intermitting pyrexia, and a loud endocardial murmur. The cause of his illness was obscure during life, but he died gradually of asthenia. After death gummata were found involving the cranial and other bones. There were striae in the cornea and other evidences of syphilis, and abundant evidence of acute recent endocarditis, and a generalised endarteritis. This case is referred to at greater length in the "Clinical Journal," Dec. 1st, 1897.

than 35 or 40 when attacked by endocarditis for the first time, (vi.) Heredity is an important predisposing factor.

The *Diagnosis* of Benign Endocarditis has been referred to above (under the Constitutional Symptoms); and it is not usually difficult. It is most important, however, to distinguish the two forms of endocarditis, as they differ so widely in their duration and fatality. Malignant or Ulcerative Endocarditis differs clinically (1) in the greater severity of the constitutional symptoms, which may present all the features of septicæmia or of the typhoid state; (2) in the wide range of the temperature in the course of twelve or twenty-four hours, and the occurrence of severe rigors and sweats; (3) in the invariable occurrence of systemic emboli, which may be of an infective character. When, however, malignant endocarditis supervenes on a previously damaged heart the diagnosis may become extremely difficult (see below).

The *prognosis*, though the malady may last for many weeks, or even months, is favourable as regards life, but the damage to the cardiac valves is generally permanent, and then the prognosis turns on many important considerations (§ 53).

*Treatment* should be directed primarily to the disease of which endocarditis is a complication—salicylate of soda, for instance, for rheumatic fever, though this drug has no control over the cardiac lesion. *Perfect rest*—hardly allowing the patient to turn in bed—is absolutely essential. This not only favours the subsidence of the inflammation, but prevents the violence of cardiac action which separates the fragments from the valves and leads to embolism. Aconite is of great value to slow and steady the heart. In this and other respects the treatment is much like that of pericarditis (§ 41); though the local treatment has less effect in endocarditis. Stimulants and digitalis are indicated only if the heart's action is very weak and irregular, and they should be given with great caution, for fear of stimulating the heart too much and promoting embolism.

§ 42a. **Ulcerative or Malignant Endocarditis** [Synonyms: Infective Endocarditis, Multiple Systemic Embolism, Arterial Pyæmia (Wilks)]. In this form the endocardium is much more seriously affected, and the inflammatory products larger and more virulent.<sup>1</sup>

<sup>1</sup> On the valves these may result in rents or perforations. In other places they invade the muscle, and may form abscesses or aneurysmal dilatations of the heart. The disease is



It seems doubtful if the disease ever occurs as a primary affection. It is usually a complication of such diseases as Pneumonia, Erysipelas, Septicæmia, Meningitis, Gonorrhœa. Dysentery (in order of frequency),

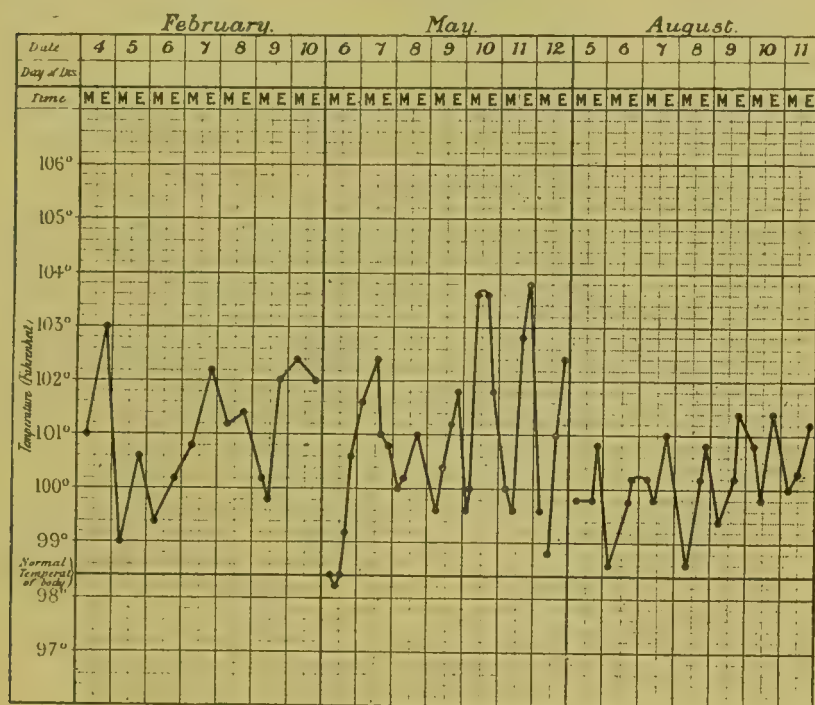


Fig. 15. Chart of Malignant or Ulcerative Endocarditis, taken from a case recently seen with Dr. Owen Pritchard, in which exhaustive inquiry failed to reveal any primary disease. The patient was a single lady, ætat. 39, whose previous history pointed to C. V. D. since the age of 29. Otherwise she had always enjoyed good health up to November, 1898, when the present illness came on gradually. At first it was mistaken for Influenza, and afterwards, when the temperature did not subside, for typhoid. Extreme lassitude, great breathlessness on movement, palpitation and pallor were the leading symptoms, with profuse and frequent perspirations at night, and at other times, occasional rigors. These symptoms, with the remitting temperature, specimens of which are shown in chart, continued throughout the illness, which lasted until August, 1899, when she died. In February, 1899, after three weeks' severe headache, her doctor told her she had optic neuritis in r. eye, and about the same time the r. arm became partially paralysed and completely pulseless. Later on the l. arm was similarly affected. Both recovered. In June, 1899, she lost the power of speech for several weeks, and her doctor told her she had had a "stroke." In July, 1899, I saw her with Dr. Pritchard, when she had a double murmur all over the cardiac area, albuminuria, and the other symptoms above noted. No autopsy was obtained.

or of the puerperium. It is met with more rarely after Chorea, Scarlatina, and Rheumatism, diseases in which simple Endocarditis is so common: and with extreme rarity after Tuberculosis, Diphtheria, and Variola. There is a marked predisposition for the disease to attack a heart which is already the seat of chronic endocarditis. (1) The constitutional symptoms vary considerably, but are usually very grave, comprising

now known to arise from several different kinds of general septic infection. The most common are Pneumococcus, Streptococcus, and Staphylococcus; it can also be produced by the Gonococcus. On this account some have suggested a name for this disease, *Streptococcus* (or some other) *Infection with Endocarditis*. (Washbourne and others, "Discussion on Infective Endocarditis," Path. Sec. B.M.A., *Lancet*, August 12th, 1899, et seq.)



intense anæmia, great prostration, and, in the (a) *Typhoid variety*, the early supervention of somnolence and muttering delirium. In the (b) *Septic variety* (such as arises with acute necrosis, the puerperium, or an external wound) the mind remains quite clear to the end, but rigors and sweats are prominent, simulating ague or pyæmia. The spleen is usually enlarged, and petechial rashes are fairly frequent. The fever is high, and may be continuous, but it more often runs an irregularly intermittent course, which may extend over weeks or months. (2) Generally there is a *cardiac murmur*, but a careful examination may be necessary, and occasionally—in cases free from old valvular mischief—there is none; so that the occurrence of embolism may be the first symptom to draw attention to the heart. (3) The *emboli* may be simple blockings of an artery, as in Simple Endocarditis, but they may become abscesses, which in turn form sources of septic infection in the lung and elsewhere (symptoms of embolism, see p. 73).

*Course and Varieties.* The severity and duration of the disease vary widely. Those cases coming on without previous cardiac mischief usually run a rapid and acute course of 5 or 6 weeks. Varieties (a) and (b) *vide supra*. (c) The *Cardiac group* (Bramwell)—those in which previous C. V. D. exists—run a prolonged and usually chronic course of many months, up to a year or more; rigors are often absent, and it may be very difficult to decide if Malignant Endocarditis is present or not. Between these extremes every grade is met with, but in the end the disease is almost always fatal. (d) There are certain aberrant forms marked by the predominance of such symptoms as jaundice, diarrhœa, parotitis, profuse sweatings, various eruptions, or pyrexia of a continued type.

The *diagnosis* from Enteric, Ague, Acute Miliary Tuberculosis, and Pyæmia may be very difficult. An intermitting or remittent temperature, rigors, petechiæ, pallor of the face, and the varying character of a cardiac murmur, are points in favour of Ulcerative Endocarditis. In *Enteric* the onset of the fever is gradual; rigors and sweats rare or absent. The inefficacy of Quinine serves to differentiate it from *Ague*. Local lung symptoms aid the diagnosis of *Acute Miliary Tuberculosis*. In *pyæmia* the cause is probably apparent, and the rigors and sweatings are more frequent; but the resemblance is sometimes so close that Wilks proposed to call the disease "Arterial Pyæmia" (see also Chap. XVIII.).

*Treatment* must be conducted on the same general lines as that of the benign variety (*q.v.*), the precautions as to rest and stimulants applying, if possible, with greater force. On theoretical grounds it would be well to administer abundance of nutriment, and to try antiseptic remedies such as quinine in full doses, antipyrin, antifebrin, sulphocarbolates, guaiacol, and the like. But recent researches (footnote, p. 74. *ante*) lead us to hope that an antitoxic serum may be available in the near future. Cases of recovery by the use of an antistreptococcic serum have been reported.<sup>1</sup> The difficulty lies in the variety of septic infections (pneumococcus, streptococcus, etc.), any of which are capable of producing malignant endocarditis. The physician must therefore first of all identify which of these various infections is in operation in the case before him. This can be done partly by an examination of the clinical history; partly by making cultures and experiments with the blood of

his patient (see Chapter on Bacteriology). When once the virus has been identified it must rest with the pathologist to provide the corresponding antitoxin.

There are two other, *rarer*, acute heart diseases in which the area of præcordial dulness is not necessarily increased—(III.) Paroxysmal Tachycardia and (IV.) Angina Pectoris. These are paroxysmal disorders WITHOUT ELEVATION OF TEMPERATURE.

§ 43. *The patient is suddenly seized with an attack of "palpitation," but WITHOUT ANY DEFINITE PAIN IN THE CHEST—the disease is probably PAROXYSMAL TACHYCARDIA (Neuro-Palpitation).*

(III.) **Paroxysmal Tachycardia** (Idiopathic Tachycardia, Heart hurry, neuro-palpitation, hysterical or nervous palpitation) is a term somewhat loosely applied to signify a quick pulse, but, unlike bradycardia, (slow-pulse), which has no meaning in itself, the term tachycardia should only be employed to denote a special disease with characteristic symptoms. Tachycardia proper, or paroxysmal tachycardia, is now a recognised, though not very common, condition. It consists of a series of paroxysms coming on at intervals, with abrupt onset, lasting for a few minutes to several hours, during which the rapidity of the pulse may amount to 200. It returns to a normal rate just as abruptly as it started. Sometimes there is no subjective distress, but it may be accompanied by a sense of constriction and suffocation, and the attack may commence with a "sinking" in the epigastrium. The face wears an anxious, terrified look, and the patient may complain of "flushing" of the general surface, with a feeling of heat or "pins and needles" all over. This is usually followed, in all the cases which I have seen, by pallor of the skin, a feeling of coldness of the extremities, with tremor, or actual shivering. Such are the symptoms of a typical attack of Paroxysmal Tachycardia; though the details may vary in different patients. The attacks return at varying intervals (days, weeks, or months); and are often determined by some emotional cause.<sup>1</sup>

The *Diagnosis* from Angina Pectoris is given under that affection (§ 44).

*Causation.* The disease is not limited to any special period of life in women; it may occur at any time from childhood to late middle age. In men, it occurs mostly from 15—25 years. No cause has been

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<sup>1</sup> The following may be quoted by way of illustration. In May, 1887, I was hastily summoned to one of the nurses in the Infirmary who had, two hours before, witnessed for the first time in her life the death of a patient. She was a healthy young woman of 25, in whom there had been previously no manifestations of hysteria. The solemnity of the scene in which she had just played her part was well calculated to have a very powerful emotional effect upon a novice, and she thereupon burst into a flood of tears. From this she recovered sufficiently to play the organ for prayers, but in the middle of the service, she was seized with violent palpitation of the heart, accompanied by a pain over the apex, a sensation of "pins and needles" down the arms and legs, and a sense of impending suffocation. I found her in a state of collapse and general tremor, and unable to remember what had happened. The pulse was beating 120 per minute, respiration sighing, and the surface of the body and limbs pale, cold, and covered with a profuse cold perspiration, having previously been, I was told, suffused with marked general redness. There were no physical signs of cardiac or other visceral disease. I administered 30 gr. of bromide of potash, 15 of chloral, and 3 ℥ liq. strychnine. She gradually rallied, and presently sunk to sleep; and the next morning she was herself again.

discovered either in the heart or other organs. At present it is supposed that the disease is due to a disordered nervous condition, either in the vagus or in the sympathetic, probably the former.<sup>1</sup> An attack may be brought on by mental or physical strain in those who are subject to the disease. Prof. Allbutt says it is not a mere incident in neurasthenia;<sup>2</sup> but the case I have narrated in the footnote on the preceding page was associated with many of the symptoms of that malady, and I have known many other cases of tachycardia similarly associated. It may also be associated with hysteria.

The *prognosis* depends upon the frequency and duration of the attacks. The condition, it would seem, can only be temporarily cured, though patients may live for years. Dr. H. C. Wood cites a case of a physician who suffered from it for 43 years. In the emotional variety recurrence of the attacks may be prevented by avoiding the cause.

*Treatment.* For an *attack*, a full dose of digitalis in brandy and water is recommended by some; others recommend that the patient should forcibly close the glottis and make a strong expiratory effort. Some patients obtain relief by strong coffee, or by iced water. Ammoniated tincture of valerian is very efficacious. Tincture of sumbul, spirit of ether and of chloroform, sal volatile, and bromide of ammonium are also useful. A seizure of this kind may frequently be cut short or averted if the patient can be induced to take some form of muscular exercise.

In the *intervals*, regular occupation, the avoidance of the various causes which are known to produce an attack, and attention to the general health are advisable. Sodium salicylate and bromides relieved a case of Dr. Allbutt's with gouty family history. Probably bromide in some form is the most useful medicine.

IV. *The patient, probably a male, at or past middle life, is suddenly seized with severe "constricting" PAIN IN THE CHEST, accompanied by a sense of suffocation—the disease is ANGINA PECTORIS.*

§ 44. **Angina Pectoris** is a paroxysmal affection in which the attacks consist of severe cramp-like pain in the region of the heart, attended by a sense of suffocation and impending death. The classical and severe type of this affection is happily very rare, but milder attacks, known as "pseudo-angina," are not uncommon.

*Symptoms.* (1) An attack comes on quite suddenly, often after some exertion (at any rate on the first occasion), and consists of acute pain in the heart, which radiates down the arms, especially the left arm. The face is expressive of the torture which the patient suffers, and, at first, is of a deadly pallor.<sup>3</sup> The limbs also are pale, benumbed, and often covered

<sup>1</sup> The clinical phenomena are best explained by the hypothesis that there is a sudden lowering of the general blood pressure, due to an equally sudden and widespread paralysis of the vasomotor nerves of the peripheral arteries.

<sup>2</sup> "System of Med.," vol. v., p. 830.

<sup>3</sup> This pallor of the surface is generally succeeded by a reddish, or sometimes cyanotic, tint of the same parts, as I have several times observed in patients at the Infirmary during the attacks [Trousseau (Clin. Lect. New. Syd. Soc., vol. iii., pp. 592, 606); and Austin (Trans. Clin. Soc., vol. iii.) have also noted this stage]. The succeeding stage of cyanosis is due to the paralytic dilatation which sometimes follows the spasm of the arterioles.

by a clammy perspiration. The patient is restless in his endeavours to assume a position of comfort. The sense of suffocation, of bodily discomfort, and of impending dissolution are extreme. The attack lasts from a few minutes to one or two hours or more, and is liable to be aggravated if the patient ventures to move from the position of ease which he may have assumed. In a certain proportion of cases death closes the scene. (2) The heart's action, when examined, is sometimes found to be unaltered, though palpitation may be complained of. In those cases which I have observed during the attack, the pulse was notably slow and feeble; and this is generally a marked feature in cases about to be fatal. It may be irregular, and in some cases it is increased in rapidity. There may be no murmur, or physical signs of any kind referable to the heart, but more usually some form of aortic valvular mischief is present (see Etiology below). (3) The mind remains clear throughout, so that the patient appreciates fully the horrors of his position. Many cases are accompanied or succeeded by a profuse flow of urine; others by profuse perspiration. Among the less frequent symptoms are tonic muscular spasms, convulsions, and vomiting. The limbs and other parts which were the seat of pain may afterwards feel "numbed." (4) In by far the larger number of cases, the patients are of the male sex, and advanced in life. Out of 88 cases collected by Sir John Forbes,<sup>1</sup> 80 were men, and 72 of these were over 50 years of age. The disease also appears to affect by preference persons among the wealthier classes of society; and, for some inexplicable reason, as Fagge and Pye-Smith point out, persons who have been possessed of unusual mental capacity.

*Varieties.* 1. When discoverable cardiac lesions are present, the disease is known as *Symptomatic Angina Pectoris*.

2. *Idiopathic Angina Pectoris* is that form in which no such organic cause can be detected.

3. *Pseudo Angina Pectoris* is a term loosely employed to designate any attack of cardiac pain and palpitation—e.g., the anginoid attacks accompanying flatulent distension of the stomach. Walshe and others describe under this term a minor form of attack of frequent occurrence, consisting of more or less severe pain, referred to the region of the heart, with palpitation, "coming on either without cause or after exertion, or through over-eating, or indigestion, or flatulent distension of the stomach, or a variety of other functional disturbances."<sup>2</sup>

4. *Hysterical Angina Pectoris* is a term sometimes employed erroneously for the condition described under Neuro-palpitation or Paroxysmal Tachycardia (§ 43).

5. *Angina Vaso-motora* is a term applied by Nothnagel to cases somewhat resembling true angina pectoris, excepting that the symptoms of vaso-motor disturbance—pallor followed by cyanosis, coldness and numbness of the extremities—predominate over the symptoms referable to the chest.<sup>3</sup>

*Diagnosis.* 1. It is important to distinguish the different forms of

<sup>1</sup> Quoted by Hilton Fagge *loc. cit.*

<sup>2</sup> Walshe, "Diseases of the Heart," 4th ed., p. 209.

<sup>3</sup> *Angina Pectoris Vaso-motora*; Deutsch Arch. Klin. Med., III. As Fagge and Pye-Smith point out ("Prin. and Prac. Med.," 2nd ed., vol. I., 912), in these cases of Nothnagel, "the state of the peripheral circulation seems to be very similar to that which in other patients leads to paroxysmal hemoglobinuria," and this Angina is brought into line with other neuro-vascular disorders.



"Anginoid attack," grouped under "*Pseudo-Angina Pectoris*," from true Angina. Among the features which sometimes enable us to distinguish Pseudo-Angina from the graver form of disease are the following: (i.) they come on at any time of life, whereas true Angina is confined to persons of the male sex over 45 years of age; (ii.) they may occur in either sex, the hysterical form being specially liable to affect young females; (iii.) they may come on spontaneously, without previous exertion (though this is not constant); (iv.) they often appear after meals, and are nearly always associated with some gastric derangement, such as dilated stomach, flatulence, etc.; (v.) the pulse of Pseudo-Angina is usually rapid and regular, never slow; and the sounds and boundaries of the heart are normal. Nevertheless, Pseudo-Angina, like true Angina, may or may not be associated with cardiac lesions. (2) Attacks of *Paroxysmal Tachycardia* are not difficult to differentiate from true Angina, on account of the great rapidity, and the regularity, of the pulse and the normal arterial tension. (3) *Biliary Colic* has occasionally to be diagnosed from angina, but here the patient advanced in years is usually of the female sex, and the condition is speedily followed by jaundice. (4) The diagnosis from the other causes of precordial pain has already been given (§ 24).

*Etiology.* The immediate cause of an attack is usually some undue exertion. After death it is said that no structural disease of the heart and arteries may be found, though far more frequently the heart walls are found to be degenerated, flabby, or fatty, with or without other changes in the cardio-vascular system.

*Anatomical Lesions.* It is very difficult to believe that true angina can occur without organic changes either in the heart or the arteries, or in both. It is very easy to overlook localised degenerated patches in the heart such as Kernig described; and as regards the arteries, I have elsewhere pointed out the ease with which important granular changes in the middle coat (the functionally active tissue) of the arterioles may remain undiscovered, especially when acid-orcein has not been employed as a stain.

Among the anatomical lesions which may be found after death in fatal Angina Pectoris are the following: (1) Fatty, or fibroid, or granular degeneration of the heart muscle is said to be the most frequent. (2) Aortic valvular disease, especially stenosis; mitral disease is rare. (3) Advanced atheroma or calcification of the aorta. (4) Aneurysm or dilatation of the aorta, especially of the root, within the pericardial sac. (5) Atheroma of the coronary arteries, calcification, or some other disease of these structures; and this may in some cases lead to embolism or thrombosis, and thus to a more or less localised degeneration of the cardiac muscle (Kernig). (6) Arterial sclerosis (using that term in its widest sense to indicate any thickening and rigidity of the arterial walls). (7) Gumma of the heart wall, in which circumstances the patient may be young. Dr. Sidney Phillips (*Lancet*, 1897, vol. i., p. 223) has collected a valuable series of cases, and from these it would appear that if small and situated elsewhere than in the l. ventricle, a gumma of the heart may give rise to no symptoms, until by its growth it produces sudden death. It is not therefore possible to diagnose cardiac syphilis with certainty. Its existence, however, may be suspected in presence of angina pectoris and a rapid irregular pulse, especially if these occur in a person under middle age, and if no other cause for these symptoms can be made out. If these be present, even in a slight degree in syphilitic persons, they should be regarded with grave apprehension; and the disappearance of these symptoms under antisymphilitic treatment, renders the diagnosis highly probable.

Angina Pectoris has been regarded as the result of disease of the coronary arteries, but it is now generally believed to be due to a sudden demand for increased effort on the part of a damaged heart. In, at any rate, a certain proportion of cases this sudden demand consists of an abrupt increase in the peripheral resistance by contraction of the peripheral arterioles. It appears that for the production of the attacks of angina the combination of these two factors is necessary. Neither of these can alone produce a paroxysm; for, as Broadbent ("*Heart Disease*," p. 303) points out, high arterial tension is extremely common, alone; so also is a degenerated heart-wall—yet angina is rare. When, however, the two are present in combination, a third or determining cause (e.g., some unusual exertion, or a further increase in the blood pressure) supervening suddenly, may produce an attack of angina. For purposes of treatment it is important to note that relief may be obtained by diminishing the peripheral resistance—i.e., by vaso-dilators.



*Prognosis.* True Angina is an extremely serious condition. The patient may die in a paroxysm. The attacks are sure to return, though this may not happen for some years. The existence of a cardio-valvular lesion, which is usual, does not materially modify the prognosis; the condition of the cardiac wall is our best guide to the probable course of a case (vide prognosis of C. V. D.).

*Treatment.* (a) *For the attacks.* Amyl nitrite 3 to 5 ℥, inhaled, generally gives prompt relief, a method of treatment for which we are indebted to Lauder Brunton. Sufferers should carry about with them glass capsules containing this quantity, which can be broken into the handkerchief.<sup>1</sup> The remedy hastens the advent of the second stage of arterial dilatation, and the attack passes off. For a more lasting effect, nitroglycerine may be given internally,  $\frac{1}{100}$  drop every 1 to 4 hours, in tabloids, pushed by tolerance of  $\frac{1}{10}$  drop. All the nitrites have a similar action in dilating the peripheral arteries; and lately, advantages have been claimed for Erythrol-tetra-nitrate,<sup>2</sup> in that its effects are more permanent. 1 grain administered in 1 drachm of absolute alcohol suitably diluted, being said to have effects lasting 4 or 5 hours. It is reported to have relieved cases in which other remedies have failed. A hypodermic injection of a full dose of morphia, if the last-named remedies are not at hand, generally gives some relief; and in very severe cases chloroform inhaled to complete anæsthesia has been recommended. If this be combined with a dose of morphia, its effects become more prolonged. In some cases of "Angina Vaso-motoria" warm baths gave great relief. I have not tried this treatment in cases of true angina, but judging from the good effects I have observed in other conditions of vascular spasm, this method of treatment would be very efficacious in cases where movement is not harmful to the patient. Mustard plasters and warm fomentations to the epigastrium may be tried, either during or between the attacks. In two cases of somewhat severe pseudo-angina, I found that the following draught carried by the sufferer in his pocket and taken at the outset of an attack was attended by prompt relief: tinct. lobeliae æth. ℥xx.; spiritus etheris, ℥xx.; liquoris morphine, ℥xx.; aquæ chloroformi, 1 oz.

(b) *Between the attacks.* It follows from the above remarks on the etiology, that the indications for treatment lie in two directions, to relieve excessive tension or any tendency to vascular spasm, and if possible to restore the damaged heart. If the main element of the case is cardiac enfeeblement, this should receive our special attention, on the lines mentioned elsewhere (Treatment of C. V. D.). If, on the other hand, the peripheral resistance tends to be excessive, our treatment should be directed to reduce this. The pulse should be examined many times, and under different conditions, during the day, and if the tension is excessive, much may be done, even though the arteries be diseased (§ 64, high tension). Erythrol-tetra-nitrate and nitro-glycerine are here again valuable remedies, not only to relieve but to prevent the occurrence of the attacks, and these may be combined with various cardiac tonics, such as iron, nux vomica, and especially arsenic. Digitalis and stimulants may be administered on the same principles as in cardiac valvular disease.

<sup>1</sup> This remedy seems to lose its effect when preserved in the ordinary way in a bottle.  
<sup>2</sup> Bradbury, *B. M. J.*, 1895, II. 1213; and Geo. Oliver, *B. M. J.*, 1896, Vol. I., p. 1433.

Much may be done by regulating the mode of life, and avoiding those things which are known by experience to induce the seizures. When, as usual, attacks are brought on by exertion, complete repose of mind and body must be strictly enforced. Other determining causes met with are exposure to cold, indigestion, dilatation of the stomach by too hearty meals, a sudden alteration of posture by the patient. Such conditions must be avoided, and so also any unnecessary or sudden exertion or emotion.

#### CHRONIC AFFECTIONS OF THE HEART AND PERICARDIUM.

§ 45. **Classification.** CHRONIC disorders of the heart and pericardium may follow an acute attack of the conditions described in the previous sections, as for instance when chronic valvular disease dates from an acute endocarditis which has complicated rheumatic fever or scarlatina in early life. But a considerable proportion of the disorders which affect the heart are chronic from the beginning, they start insidiously, and are unaccompanied throughout by constitutional or other marked symptoms.

For clinical purposes, the chronic, like the acute disorders of the heart and pericardium may be divided into those attended by enlargement of the area of dulness and those *not necessarily* so attended.

a. CHRONIC Diseases attended by ENLARGEMENT of the area of præcordial dulness.

I. Cardiac hypertrophy.

II. Cardiac dilatation.

III. Hydro-pericardium.

IV. Congenital heart disease (rare). *Mem.*—Aneurysm, and other Mediastinal Tumours. (See footnote to table on p. 64.)

b. CHRONIC Diseases NOT NECESSARILY attended by ENLARGEMENT of the area of præcordial dulness, the diagnosis of which depends mainly on auscultation.

I. Valvular disease.

II. Fatty heart.

It is important to bear in mind that Valvular Disease, though not *per se* giving rise to an enlarged area of præcordial dulness, is so often associated with hypertrophy or dilatation that it is frequently attended by enlargement of the præcordial dulness.

**Method of procedure.** It will be remembered that the routine examination of the heart consisted of—1st, Inspection ;

2ndly, Localisation of the apex ; 3rdly, Percussing the præcordial dulness ; and 4thly, Auscultation. The student should bear in mind the various *fallacies* which may give a false impression of cardiac enlargement, and also those conditions, such as, emphysema, which obscure an enlarged heart (§ 36). If the area of dulness is NOT INCREASED, turn to § 50.

GROUP a. The patient complains of some of the subjective symptoms pointing to **chronic cardiac disorder** ; and, on examination of the heart, the **area of dulness is found to be increased**, chiefly in the transverse direction, the disease is probably—**Hypertrophy, Dilatation, or Hydropericardium.**

I. *The APEX BEATS BELOW its normal position, the impulse is FORCIBLE and heaving. On auscultation the first sound is DULL and prolonged. . There is HYPERTROPHY OF THE HEART.*

§ 46. **Hypertrophy of the Heart**, and the dilatation which so frequently accompanies or follows it, are certainly the commonest conditions which produce an increased area of præcordial dulness.

*Cardiac Hypertrophy* is an increase of the muscular substance of the heart, and its weight, which is normally about  $8\frac{1}{2}$  ozs. in women, and  $9\frac{1}{2}$  ozs. in men, may be increased to 10 or 12 ozs., and on rare occasions to 15 or 20. Its *signs* are as follows : (1) The increase in the præcordial dulness is in a transverse direction—towards the l. if the l. ventricle be hypertrophied, towards the r. if the r. ventricle. (2) The apex beats below its normal position. (3) The impulse is unduly forcible, heaving, or thrusting. (4) On auscultation, the first sound is muffled, less audible, and prolonged. The pulse is firm, strong, and bounding.

*Symptoms* may be altogether wanting if the hypertrophy accurately compensates for the obstruction in the circulation which has caused the hypertrophy. The patient may indeed be unaware of any cardiac disorder. But generally, on inquiry, he will complain of a “thumping” in his chest, and “throbbing” in his head, occasionally of breathlessness and præcordial distress.

*Etiology.* Cardiac hypertrophy—and the dilatation which often accompanies it—is always the result of some obstruction in the circulation, either in the lungs (such as bronchitis and emphysema) or in the general circulation (such as cardiac valvular disease, or

arterial thickening). It is an illustration of the physiological law that increased use leads to increased growth. The part of the heart which chiefly undergoes hypertrophy will depend on the position of the obstruction; and the signs met with in addition to those above mentioned will vary accordingly. Thus, there will be 3 sets of symptoms—(a) Signs of hypertrophy of the heart; (b) signs of enlargement of the cavity specially involved; and (c) signs and symptoms of the cause which is in operation. The following causes will be more readily understood by consulting Fig. 16 (p. 94), and it must be remembered that the enlargement is never, in actual practice, strictly limited to one chamber of the heart.

(a) HYPERTROPHY OF THE L. VENTRICLE is indicated by enlargement of the area of dulness, chiefly towards the l.; the apex beats *below* and to the l. of its normal position, the pulse is strong unless modified by the presence of a valvular lesion, and the carotids may be seen to pulsate. This condition may arise under nine different causes:—

(i.) *Mitral regurgitation*, in which case there would be a systolic apical murmur and the other features given in § 50.

(ii.) *Aortic stenosis or regurgitation*, which may be recognised by a basal murmur of systolic or diastolic rhythm and other characters given in § 50. The hypertrophy resulting from regurgitation may be greater than that due to any other cause (cor bovinum of the ancients). The heart may weigh in these cases 20 to 30 ozs. or more. In regurgitant lesions, a certain amount of dilatation always accompanies hypertrophy; and the condition is then known as "eccentric" hypertrophy. In these cases the dilatation is compensatory and produces no untoward symptoms. True, or as it is called "concentric," hypertrophy, unaccompanied by any dilatation, is only met with in stenosis, especially aortic stenosis.

(iii.) *Aneurysm of the aortic arch*, if unattended by valvular disease or renal mischief, does not *per se* cause cardiac hypertrophy; but practically it is nearly always so attended, and thus becomes a fairly frequent cause of hypertrophy of the left ventricle. If the aneurysm involves the first half of the arch it produces marked physical signs: if the second or third part, *pressure* symptoms arise without signs (§ 56).

(iv.) Prolonged *high arterial tension*—and thus its numerous causes (§ 64)—may lead to hypertrophy of the left ventricle. It is probably in this way that Chronic Bright's disease is so frequently accompanied by hypertrophy of the left ventricle.

(v.) Widespread *thickening of the peripheral arterioles* is invariably followed, sooner or later, by a certain degree of hypertrophy of the left ventricle (arterial sclerosis, § 70).

(vi.) *Disturbed innervation*, such as attends the "irritable heart," Graves' disease, and *neuro-palpitation*, in time results in moderate hypertrophy, chiefly of the left ventricle.

(vii.) *Pericardial adhesions* are still mentioned by some authors as a cause of cardiac hypertrophy (*vide* footnote to Adherent Pericardium, § 41).



(viii.) *Cardiac fibrosis* (sclerotic myocarditis) is a rare condition, which may be associated with a certain amount of hypertrophy.

(ix.) *Increased density of the blood* is a cause of hypertrophy which is not mentioned among the lists usually given in text-books, but which, nevertheless, must be of considerable potency. It is estimated that 99 per cent. of the work done by the heart is employed in overcoming the resistance due to capillarity in the arteries and capillaries, and it follows, almost of necessity, that if the density of their fluid contents be increased the resistance will be increased proportionately.

(b) HYPERTROPHY OF THE R. VENTRICLE is indicated by enlargement of the area of dulness to the r.; throbbing and pulsation in the lower l. intercostal spaces and epigastrium (and if accompanied by dilatation, pulsation also in the veins of the neck); a violent but more *diffuse* apex beat; and, on auscultation, accentuation of the 2nd sound over the pulmonary valves. *The degree of hypertrophy present may be measured by the degree of the second and fourth of these symptoms*: in this way we measure the efficiency of compensation (§ 53).

It may be due to three important causes:—

(i.) *Pulmonary diseases* attended by obstruction in the pulmonary circulation, of which *bronchitis* and *emphysema* are certainly the most frequent. This combination, a very common one, is identified by a history or evidence of lung mischief (§ 97).

(ii.) *Mitral stenosis* is the next most common cause, and should be borne in mind even in the absence of a presystolic murmur (§ 50).

(iii.) *Mitral regurgitation* is followed by hypertrophy of the r. ventricle, due to the backward pressure through the lungs.

(c) HYPERTROPHY OF THE L. AURICLE is always attended by dilatation. It is a difficult condition to detect; because the palpable and visible pulsation in the 3rd l. interspace when present, though due to this cause, may admit of other explanations.

It may arise in *mitral regurgitation*, but its chief cause is *mitral stenosis*. In the latter condition, palpation generally reveals a thrill over the apex, and careful auscultation may detect the presystolic or mid-diastolic murmur (§ 50).

Hypertrophy of the R. Auricle is not recognisable clinically.

(d) EXTREME HYPERTROPHY OF BOTH AURICLES AND VENTRICLES arises in Congenital Heart Disease; although the valvular lesions are mostly on the r. side.

*Obscure causes.* If, in a given case of cardiac hypertrophy, careful examination reveals no valvular mischief, and no obvious cause can be made out, the physician should always suspect OBSCURE AORTIC ANEURYSM, OBSCURE RENAL DISEASE, or wide-spread ARTERIAL THICKENING.

*Idiopathic Hypertrophy.* The heart as a whole continues to increase slightly in weight and size to an *advanced age*, though, as a rule, there are no clinical evidences of this normal growth. *Excessive muscular exercise*, either athletic or laborious, may produce hypertrophy; and in support of this statement it may be mentioned that the normal



increase, with age, is more noticeable in men than in women. Apart from these circumstances of age and exercise, *idiopathic hypertrophy* is practically unknown, and it is possible that in many cases of supposed idiopathic hypertrophy the cause may have been overlooked.

*Prognosis and Treatment.* Cardiac Hypertrophy is in itself essentially a conservative process for some condition which causes obstruction in the circulation. It is nature's method of compensating for the obstruction, and it is well to promote it up to a certain point.

1. *If the cause be removable*, the prognosis is favourable. Our treatment in such cases should therefore be directed to the removal of the cause—*e.g.*, high arterial tension, which can be reduced.

2. *If the cause be not removable*, the prognosis of the case depends on our being able first to maintain the compensatory hypertrophy, and secondly, to relieve the heart of part of its work, so that the hypertrophy does not go beyond what is necessary. To accomplish the first we should endeavour to promote the general nutrition by tonics and hygienic measures, and by regulating the emunctories with mild saline laxatives, and similar remedies. In order to relieve the heart of part of its work and to aid the systemic circulation, baths, massage, passive and active movements, are of the greatest use (see § 54).

3. *The existence of cardiac hypertrophy* adds an element of risk to a person's life in three ways. In the first place, hypertrophy infallibly indicates that there is obstruction somewhere in the circulation, and this, whatever it be, is in itself an injury to health, and may shorten life. Secondly, a far more important consideration is the increased liability to cerebral hæmorrhage, and vascular rupture elsewhere, which cardiac hypertrophy entails. Thirdly, high arterial tension is an almost invariable accompaniment of cardiac hypertrophy, and this causes a continual strain upon the peripheral vessels, which results first in arterial hypermyotrophy and later in arterial sclerosis, the serious consequences of which are indicated elsewhere (§ 70).

II. *The area is increased*; the position of the APEX is INDEFINITE; the impulse is diffuse and wavy. On auscultation the first sound is short and sharp. The disease is CARDIAC DILATATION.

§ 47. **Cardiac Dilatation** (one form of "Cardiac failure") is an indication that the heart is giving way, or "failing" to keep pace with the extra demand made upon it by reason of some

obstruction in the circulation. In ordinary circumstances the heart first hypertrophies, then dilates; and that is why hypertrophy and dilatation are usually associated. But if the nutrition of the body or of the heart is faulty the heart begins to dilate from the outset, without any preliminary hypertrophy.<sup>1</sup>

The *physical signs* of cardiac dilatation resemble those of hypertrophy in several ways; and, like it, (1) the area of dulness is increased chiefly in a transverse direction, to the r. or to the l., according to the side of the heart which is dilated. But there are three important features specially belonging to dilatation—(2) The apex-beat is wavy and diffuse, as well as being displaced downwards; it may be so feeble as to be hardly perceptible. (3) On auscultation, the first at the apex sound is clear and sharp, resembling the normal second sound in character. Murmurs may be present from co-existing valvular disease, but a *systolic murmur*, the “murmur of dilatation,” may sometimes be heard apart from actual valvular disease, because the auriculo-ventricular orifices, by reason of the dilatation, allow a reflux of the blood. (4) The pulse is feeble, rapid, irregular, and sometimes intermittent (see also Table IV.).

TABLE IV.—DIAGNOSIS OF TYPICAL CARDIAC HYPERTROPHY FROM TYPICAL DILATATION.

Apex-Beat and Impulse; Displaced in both.	Percussion.	Auscultation.	General Symptoms.
<b>Hypertrophy.</b> Forcible, heaving, thrusting; below and to l. of normal (l. ventricle); in epigastrium (r. ventricle).	{ L. V. :—area increased transversely to the l. R. V. :—area increased transversely to the r. }	{ Sounds muffled, prolonged, and forcible. }	{ May be absent; or symptoms of high arterial tension. }
<b>Dilatation.</b> Feeble, irregular, undulatory diffuse. If r. ventricle—pulsation in the epigastrium and veins of the neck.	{ L. V. :—area increased transversely to the l. R. V. :—area increased transversely to the r. }	{ Systolic murmur at apex, at one stage. Systolic murmur in tricuspid area, at one stage. } Sounds sharp and clear.	{ Dyspnoea, cough, cyanosis, and other signs of lung congestion. Dropsy, scanty high-coloured albuminous urine, enlarged liver, ascites, and other signs of congestion of organs. }

Towards the end, when cardiac failure is extreme, Fœtal Rhythm, Gallop Rhythm, and “Delirium Cordis” may occur. In Fœtal Rhythm the

<sup>1</sup> Cardiac enlargement is revealed upon a carefully made skiogram (Fig. 13), but we are not yet in a position to decide from this the relative proportion of hypertrophy and dilatation. This important question still depends for its decision upon the other symptoms present.

long and short pauses are almost identical, so that the first and second sounds can scarcely be distinguished; in Gallop Rhythm there is rapidity of action, together with a distinctly reduplicated second sound. In Delirium Cordis the heart is so rapid and so irregular that it is practically impossible to make out the relations of sounds and murmurs.

It is, however, by the presence of certain *symptoms* that the existence of cardiac dilatation (or failure of compensation) generally becomes manifest. In Hypertrophy, as we have seen, there may be no symptoms at all; but with Dilatation the patient complains of (1) *Heart symptoms*, such as breathlessness on little or no exertion, palpitation, and præcordial distress. (2) There may also arise a number of *symptoms referable to other parts*, in consequence of the delay in the circulation, such as anasæra, ascites, and symptoms of congestion of the lungs, liver, and kidneys. These will be described under Cardiac Valvular Disease; where the means of detecting which cavity is chiefly involved is also given. (See also Table IV.)

The *Causes of Cardiac Dilatation* are of extreme importance as bearing on the prognosis and treatment of cardiac valvular disease and other circulatory disorders. The *clinical conditions* which produce Dilatation are practically identical with those which produce cardiac hypertrophy (pp. 83—5) when they are persistent and are *associated with some condition which impairs the nutrition of the heart* (see below). Undoubtedly the two commonest causes of Cardiac Hypertrophy and Dilatation are CARDIAC VALVULAR DISEASE, and CHRONIC BRONCHITIS WITH EMPHYSEMA, and these are the possibilities which should first suggest themselves to the mind in a case where dilatation is evident. The former will be fully discussed in the following section.

*Chronic Bronchitis* with its usual accompaniment of Emphysema produces in time a dilated *Right Ventricle*. This latter is recognised by two very characteristic local signs in addition to the breathlessness, etc., above mentioned—viz., (i.) Epigastric pulsation, and (ii.) pulsation in the jugular veins. The clinical picture presented by this frequent pathological combination is very characteristic—the florid face and plethoric build; the easily excited breathlessness and constantly recurring cough—enable us to recognise the condition almost at a glance. The subject will be more fully discussed under C. V. D. (p. 95).

The *Essential or pathological causes* of Dilatation may be arranged under 4 headings:—

a. Any condition which persistently *prevents the complete emptying of the cavities of the heart* (see causes of hypertrophy) will produce compensatory hypertrophy with dilatation which will be *exactly proportional* to the increased resistance in the circulation; provided none of the circumstances mentioned under (b) (c) or (d) below are also present. If any of these circumstances are in operation, dilatation or failure may be initiated without previous or accompanying hypertrophy. Moreover, the supervention of any of these in the course of a cardiac case may at once disturb a well-balanced compensatory hypertrophy, and serious symptoms may immediately appear.

b. Any *failure of a general nutrition*, or vitality, may entail a weakened cardiac wall, which will, perhaps, yield in presence even of normal circulatory conditions; such, for instance, as exposure, insufficient food, alcoholic excesses, old age, various fevers (especially Rheumatic fever, Typhus, Typhoid, and Malaria), various blood conditions (such as pernicious anæmia, scurvy, chlorosis, leukæmia, etc.), and various cachectic conditions (such as syphilis, tubercle, and cancer). See Causes of Pyrexia and of Anæmia (Chapters XVIII. and XIX.).

c. *Local impairment of the nutrition of the heart-wall* may result in dilatation without hypertrophy, even with normal circulatory resistance. Myocarditis, for instance, and the conditions which accompany peri- and endocarditis (which lead sometimes to acute dilatation); or the more gradual degenerations which ensue on sclerosis and other diseases of the coronary arteries; or fibroid and other degenerations of the cardiac wall (see Fatty Heart). Prolonged fatigue may also act locally by over-taxing the heart muscle. Any of these may upset the balance of a well adjusted hypertrophy.

d. Any *sudden strain on an apparently normal heart* may produce acute dilatation. Thus, severe and sudden grief, fright, or anxiety, may damage the heart through its nervous apparatus; and severe muscular exertion in athletes or others who have not had any previous training may cause the heart to give way and dilate. Instances of the latter are met with in hill climbers who are "out of form," and others who take sudden and unaccustomed exercise. Breathlessness may date from incidents of this kind, from which the patient may never, or only with difficulty, recover.

The *Prognosis and Treatment* of Cardiac Dilatation is fully dealt with under C. V. D. (§ 50).

III. *The area of dulness is INCREASED UPWARDS, and its shape is pyramidal, with the point upwards. The apex-beat is raised, and the impulse is weak and undulatory. On auscultation the sounds are feeble. The disease is HYDRO-PERICARDIUM.*

§ 48. **Hydro-Pericardium** is a chronic effusion of fluid into the pericardium. (1) The shape of the dulness is very characteristic, being pyramidal with the narrow end upwards. (2) The apex of the heart is *raised*, and to the *r.* of its normal position, because the roof of the pericardium is raised by the fluid and takes the heart with it. (3) For the same reason, the l. margin of præcordial dulness extends *beyond* the apex-beat. (4.) On auscultation the heart sounds are distant and muffled. There may be irregularity and rapidity of the pulse, and difficulty of breathing from the impeded action of the heart and lungs.



*Etiology.* Chronic effusion into the pericardium may originate in one of three ways. (1) As the result of acute pericarditis (§ 41), of which a history is generally, but by no means always (see latent pericarditis § 41 Ib), obtainable. Most authors draw a distinction between chronic pericardial effusion of inflammatory origin, and simple dropsy of the pericardium (hydro-pericardium proper). But the physical signs are practically indistinguishable; for the diagnosis of the former we depend mainly on the history of it having commenced as an acute affection, and on the absence of anasarca. (2) True hydro-pericardium seldom occurs excepting as part of a general dropsy due to renal or cardiac disease; and therefore the urine should be carefully examined. In these circumstances the effusion gives relatively *little inconvenience to the patient*, because it takes place so gradually that the parts have time to adapt themselves to the stretching they undergo.

(3) If hydro-pericardium be not preceded by pericarditis, or be not part of a general dropsy, new-growth (e.g., cancer or tubercle), although it is rare, should always be suspected. In these circumstances, if a little fluid be withdrawn by a hypodermic syringe (p. 70), it may be blood-stained (cancer), or contain bacilli (tubercle).

The *Diagnosis* from Cardiac Dilatation should be readily accomplished by the square, instead of pyramidal, shape of the dulness, and the heart sounds being clear and sharp, instead of muffled. Pleuritic effusion is attended by pulmonary symptoms.

The *prognosis* of hydro-pericardium depends on its causation, being favourable in cause 1; adding only a little to the gravity of the primary malady in 2; and being almost necessarily fatal in 3.

*Treatment.* The treatment of inflammatory effusion is dealt with in § 41. If part of a general dropsy our efforts must be directed to this. Counter-irritants are sometimes useful. Paracentesis should not be considered unless the cardiac embarrassment is very urgent, because of the danger of withdrawing a large amount of fluid suddenly from the pericardial sac; and because the risks of the operation are not small.

IV. *The præcordial percussion area is considerably and irregularly increased; the impulse is forcible and heaving.*

*a.* *The area is DISTORTED and somewhat SQUARE. On auscultation there is a loud murmur, probably loudest in the pulmonary area:—the disease is CONGENITAL HEART DISEASE (see below).*

*β.* *The UPPER PART of the area is increased transversely, and there is dulness OVER THE STERNUM:—it is probably an INTRATHORACIC TUMOUR. If, on auscultation, the second sound at the base is reinforced and sharp or replaced by a diastolic murmur:—it is probably*

*ANEURYSM of the first part of the ARCH OF THE AORTA (§ 56).*

§ 49. *Congenital heart disease* is another chronic form of cardiac disorder attended by increased præcordial dulness, but it is comparatively rare. There are three cardinal signs attaching to it—1. The præcordial dulness is very considerably *increased*, but in addition to this the normal shape is *distorted*, and it may extend considerably beyond the r. border of the sternum, because the commonest form of the disease results in immense hypertrophy and dilatation of the r. ventricle. 2. Palpable and sometimes visible pulsation over almost the whole of the cardiac area may often be detected for the same reason. 3. A loud, rough systolic murmur can generally be heard, loudest in the third or fourth interspace, close to the l. of the sternum. These signs in a child who has a tendency to cyanosis are almost certainly due to cardiac malformation. 4. Dyspnoea is also pretty constant, and may be either persistent or paroxysmal. The condition,



however, may remain latent for many years, until exertion or some illness reveals its existence. The diagnosis is sometimes a matter of difficulty. Other symptoms arise as the disease progresses,—thus, general cyanosis reaching a very extreme degree, greater than in any other condition; extreme coldness of the extremities; a low temperature of the surface generally (because the blood is poor in oxygen), although not of the interior of the body (Peacock); dropsy occasionally; hæmorrhages from the lungs; and symptoms of congestion of the other viscera. Dilatation of the conjunctival vessels is often observed, and clubbing of the toe and finger ends. Headache is often present, and convulsions are not unknown.

*Etiology.* Congenital disease of the heart arises under two conditions: 1. *Inflammatory affections* attacking the fetal heart in utero may lead to stenosis of the orifices; almost invariably on the r. side of the heart, the converse of extra-uterine life. 2. An *arrest of the closure*, which normally takes place shortly after birth, of either the ductus arteriosus, foramen ovale, or the ventricular septum. Whatever the cause, the commonest lesion is a narrowing of the pulmonary artery or pulmonary valves,<sup>1</sup> which probably results in the venous blood making its way through the foramen ovale, or ventricular septum, from the r. to the l. side of the heart; the septum also deviating to the l. Stenosis of the aortic orifice is much rarer, but in that case the arterial blood finds its way through the same orifices from the l. to the r. side of the heart. In either case three events happen: 1, deficient oxygenation and probably admixture of venous and arterial blood; 2, the r. ventricle takes an *equal* share with the l. in the work of the heart, and consequently it hypertrophies and dilates; and 3, the ductus arteriosus remains patent to compensate for the insufficient delivery of blood into the aorta or pulmonary artery, as the case may be. It is only occasionally possible to suggest the precise nature of the lesion during life, but this, although it is a matter of great interest, is not always of great moment.

*Prognosis.* The condition may remain latent for many years, though few marked cases survive to adult life. The prognosis is serious in proportion to the degree of dyspnoea and cyanosis, pointing to deficient aëration of the blood; and in proportion to the other symptoms of "cardiac failure" (§ 53).

The treatment is the same as that of cardiac dilatation, bearing in mind that rest is of primary importance (§ 54).

We now turn to those Chronic Heart Diseases in which the area of dulness is not necessarily increased, and which depend mainly on AUSCULTATION for their diagnosis (Table V. below).

V. *On auscultation, one or both of the heart sounds is accompanied by a blowing sound,—i.e., a MURMUR, or bruit. Pericardial friction having been excluded (§ 42), the VALVES of the heart are diseased.*<sup>2</sup>

§ 50. **Chronic Endocarditis: Cardiac Valvular Disease (C. V. D.): Cardiac Murmurs.** Disease of the valves of the heart is the commonest of all cardiac disorders; and it is revealed on auscultation by the presence of a bruit or murmur which is added to or replaces one or both of the heart sounds.

*Method of procedure.* In order to arrive at a diagnosis it will be remembered that four features must be carefully investigated in

<sup>1</sup> Some observers maintain that the primary mischief is always the non-closure of the ductus arteriosus or foramen ovale, or ventricular septum; narrowing of the aorta and pulmonary arteries being secondary. But the view above stated is the more probable, because a small leakage through one of these orifices is a by no means infrequent occurrence without symptoms during life. Some observers also maintain that there is *no admixture of the venous and arterial blood*, the cyanosis being due to deficient oxygenation.

<sup>2</sup> With certain exceptions or fallacies (such as hæmic murmurs) which will be described.

any given murmur—namely, its RHYTHM, POSITION, PROPAGATION, and CHARACTER (§ 36). The last named is relatively less important. In order to be quite sure of the rhythm of a bruit it is often convenient to place the fingers on the carotid artery whilst auscultating the chest.

A cardiac murmur may arise in three ways. It may arise outside the heart—*e.g.*, from roughness of the PERICARDIUM; it may be of HÆMIC or FUNCTIONAL ORIGIN; or it may arise within the heart from ORGANIC DISEASE OF THE VALVES (which concerns us now).

The characters of PERICARDIAL MURMURS have already been given (§ 41); and their diagnosis from endocardial murmurs (Table, p. 71).

HÆMIC or functional MURMURS are frequently heard in anæmia and in some other blood conditions (see Chapter XVIII.). They are usually single (systolic) in rhythm. They are rarely double, and never diastolic. They are usually heard loudest in the pulmonary area. A single murmur of presystolic or diastolic rhythm is a certain indication of organic disease at one of the cardiac orifices.

**Organic Murmurs** are those which are produced by organic disease of the valves (Cardio-Valvular Disease). Valvular disease may be due to several lesions (p. 102), but the commonest one in early life is endocarditis (acute or chronic), and in older persons chronic degenerative change. The effect is a thickening or puckering of the valves, which results in one or both of two conditions: (a) *Stenosis*—*i.e.*, a narrowing (*στενωω*, to contract) of the orifice which prevents the blood flowing freely through it; or (b) *Regurgitation*, in which the valve is incompetent and allows a reflux of the blood to take place from imperfect meeting and closure of the valves. The remote effect of these two conditions is practically the same—*viz.*, a retardation or obstruction to the circulation of blood through that orifice.

It simplifies matters very much that C. V. D. arising *after* birth is practically confined to the left side of the heart—*i.e.*, to the mitral and aortic orifices. Thus it happens that there are four principal cardiac lesions—MITRAL REGURGITATION, MITRAL STENOSIS, AORTIC REGURGITATION, and AORTIC STENOSIS.

TABLE V.

Differentiation of CARDIAC VALVULAR DISEASES.

		Ausculta- tion.	Appear- ance of Patient.	Pulse.	Other Symptoms special to the Disease.			
C. V. D.	Mitral. (apical murmurs).	Regurgi- tation.	Systolic murmur pro- pagated into axilla.	Florid.	Irregular, rapid and com- pressible.	Dropsy, enlarged liver and ascites, etc.	with signs of congestion of organs.	
		Stenosis.	Presystolic murmur.	Patient young.	Irregular, small, and hard.			Hæmopty- sis ; emboli.
	Aortic. (basal murmurs).	Regurgi- tation	Diastolic murmur conducted down sternum.	Sallow.	"Water- hammer," rapid and compressible.	Throbbing of arteries of neck.		with symp- toms of cerebral anæmia and anginoid attacks.
		Stenosis. <sup>1</sup>	Systolic murmur pro- pagated into vessels of the neck.	Heart lesion of the aged.	Slow, regu- lar, small and hard.	No special symptoms.		

The student should study Fig. 10, so as to thoroughly comprehend the various events which occur during one complete contraction and dilatation of the heart (a cardiac cycle). He should also bear in mind that the l. side of the heart is behind the r., and that the l. ventricle comes nearest to the surface only at the apex immediately behind, or just below the fifth rib (Figs. 11 and 12). He should also remember that a cardiac murmur is not produced *in* a diseased orifice but by the eddies in the blood stream *beyond*. For these reasons a murmur is not always heard loudest directly over the orifice diseased. The student should also consult the diagram of the circulation (Fig. 16, p. 94), and all these facts will be readily understood.

**Diagnosis of Cardiac Murmurs.**—The first thing to determine is whether a given murmur is related to the first or second sound of the heart—*i.e.*, whether its rhythm is Systolic or Diastolic—and this will form a convenient basis of classification of cardiac murmurs.

**A. Systolic Murmurs**<sup>2</sup>—*i.e.*, bruits added to or replacing the first sound— may be produced by the following causes, which are mentioned more or less in order of frequency : Hæmic conditions (see above, and Anæmia, Chapter XIX.), Mitral Regurgitation, Aortic stenosis, Aortic Aneurysm, Tricuspid Regurgitation, Pulmonary Stenosis, Congenital "milk-spot," and Cardio-pulmonary conditions.

I. In **Mitral Regurgitation** the systolic murmur is charac-

<sup>1</sup> Real Aortic Stenosis is very rare, but atheromatous roughening is very common.

<sup>2</sup> Systolic murmurs are sometimes spoken of as Ventricular Systolic or V. S. murmurs, being produced by the systole of the ventricle.



terised by, (i.) being loudest at the apex; (ii.) being propagated to the axilla, and also audible behind, at the angle of the scapula; and (iii.) owing to the resulting hypertrophy of the l. ventricle the apex is displaced downwards and outwards. There is accentuation of the 2nd sound in the pulmonary area, due to the backward pressure in the circulation. The pulse is soft, there is a characteristic florid physiognomy, and a tendency to dropsy.

*General Symptoms of mitral regurgitation* arise when there is failing compensation; and two events take place which in order of occurrence are, 1st, *dilatation of the l. ventricle and pulmonary congestion*; and 2nd, *dilatation of the r. ventricle*.

1. The symptoms of *dilatation of the l. ventricle* have been already mentioned (p. 86). *Pulmonary congestion* is revealed by laboured breathing, cough, expectoration of mucus sometimes tinged with blood, or actual hæmoptysis. The physical signs are abundant mucous râles, and sometimes scattered patches of dulness at one or both bases.

FIG. 16.—Scheme of the Circulation of the Blood. The Superior and Inferior Vena Cava (6) bring the blood back from the organs and tissues into the R. Auricle (1). Thence it passes into the R. Ventricle (2), through the Pulmonary Artery (7) into the Lungs. Returning from the Lungs by the Pulmonary Veins (9), it passes through the L. Auricle (3) and L. Ventricle (4), and is distributed by means of the Aorta (5, 6) and the Carotids (8) to the organs and tissues of the body. Notice that the blood from the liver before joining the general circulation. (From Huxley's Physiology, modified.)



*Pulmonary apoplexy* may be suspected by sudden increase of dyspnœa, accompanied by continuous hæmoptysis.

2. *Dilatation of the r. ventricle*, consequent on the backward pressure through the lungs, results sooner or later in the following symptoms and conditions.

(i.) A tricuspid *bruit* is sometimes heard (see III., p. 96).

(ii.) *Pulsation* in the epigastrium and in the veins of the neck.

(iii.) *Dropsy*, which indicates congestion of the whole venous system. Cardiac dropsy *starts and predominates in the legs, or the back*, whichever may happen to have been in the most dependent position. The skin is tense, and is very liable to be attacked by erythematous, erysipelatous, and inflammatory conditions (cellulitis, ulcer, etc.). *Ascites* in varying amount is generally present. It is often an early and prominent sign in mitral stenosis. *Cyanosis* and a general lividity of the surface are consequences of the same venous stasis. A case of mitral disease, therefore, presents a marked contrast to one of aortic disease, where the countenance is pale and sallow.

(iv.) *Engorgement of the liver* is evidenced by pain and tenderness in that region, and jaundice of the skin and conjunctivæ. The organ is enlarged, and it may extend even to the umbilicus.<sup>1</sup> Sometimes pulsation of the liver may be made out by placing one hand on the epigastrium and pressing the other beneath the back in the dorsal region.

(v.) *Indigestion, i.e.*—want of appetite, a sense of discomfort in the stomach after meals, nausea or actual vomiting, with streaks of blood, indicate congestion of that organ.

(vi.) *Albuminuria*, with high-coloured scanty urine of high specific gravity (and possibly casts in long-standing cases) points to congestion of the kidney.

(vii.) *Splenic enlargement* and tenderness are the only indications of congestion of that organ.

Ia. A MURMUR OF DILATATION, systolic in rhythm, having all the above characters, and like it (due to mitral regurgitation, may occur without definite disease of the valve, when the *l. ventricle becomes dilated*, and the muscular ring around the valve *fails to complete* the closure of the mitral valve. This condition is especially apt to occur in the aged when dilatation of the l. ventricle supervenes on hypertrophy.

<sup>1</sup> In cases of dropsy with albuminuria when we are in doubt whether the dropsy is of renal or cardiac origin, hepatic enlargement is a valuable diagnostic aid, for its presence is very usual in cardiac cases, but it is not one of the consequences of renal disease.



**II. Aortic Stenosis** is another lesion producing a systolic bruit. True stenosis of the aortic orifice is not common, but a roughness, or the presence of vegetations on the inner surface of the valves, may produce the same bruit. The latter is mostly found in old people on account of the degenerative changes, and a systolic murmur thus produced is of relatively less serious import.

This murmur is characterised by (i.) being loudest at the 2nd r. interspace; (ii.) it is conducted up to the vessels in the neck, and is audible in some cases also at the apex (Fig. 17); (iii.) it is usually harsh, sometimes musical, but in many cases it is barely audible; (iv.) the pulse is small, sustained, and slow, sometimes anacrotic (§ 62). A thrill is sometimes felt over the base of the heart.

*General symptoms* are almost wanting in aortic stenosis—other than occasional pain, pallor of the face, and faintness or giddiness—until perhaps the mitral valve, owing to backward pressure, gives way (see Mitral Regurgitation, p. 93).

The detection of aortic stenosis is sometimes as difficult as mitral stenosis, and, like it, the characteristic murmur may be absent. It may then be suspected when the patient, generally an elderly man, presents persistent dyspnoea, bradycardia, nervousness, and occasionally anginoid attacks, which are not otherwise accounted for.

**III. TRICUSPID REGURGITATION** takes place when that orifice is diseased or DILATED. Some maintain that if the valve be healthy though dilated no bruit can be heard, but certain it is that a murmur is often present in cases of confirmed bronchitis, which comes and goes under treatment, and which is not found to be attended with any marked changes in the tricuspid valve after death.

The murmur is characterised by (i.) being heard best at the tricuspid area—i.e., on the l. side of the lower part of the sternum; (ii.) it may be heard as far out as the r. nipple; (iii.) the pulse is of low tension, often irregular; (iv.) owing to the accompanying hypertrophy or dilatation of the r. ventricle the area of dulness extends to the r., and there is epigastric pulsation; and (v.) there is also pulsation of the veins of the neck, which is distinguished from the undulation seen in simple back-pressure by the fact that the pulsation is not obliterated, but is rendered more distinct, when the finger is placed on the external jugular vein.

*General Symptoms*, as above indicated (p. 94), result from tricuspid regurgitation. By far the commonest cause is Chronic Bronchitis, which thus presents a clinical picture (p. 88) readily recognised.

**IV. PULMONARY STENOSIS** is practically unknown, unless it be part of congenital malformation of the heart. This murmur is systolic in rhythm, loud and harsh, and is heard over a very wide area, but most distinctly in the 2nd left interspace.

FALLACIES IN THE DIAGNOSIS OF SYSTOLIC MURMURS. 1. *Hæmic* murmurs (Chapter XIX.) are undoubtedly extremely common, and sometimes very difficult to distinguish from C. V. D.

2. A systolic murmur audible in the *aortic area*, and having all the characters of II. above, is due not so often to aortic stenosis as to (i.) roughening of the valves in old people from *atheroma* or calcareous deposit. (ii.) Simple *incompetence* of the aortic valves may produce both a systolic and a diastolic murmur without there being any stenosis. (iii.) *Atheroma*, with *aneurysmal* dilatation of the aorta, may also produce a systolic or a double bruit; then there is a ringing 2nd sound in addition.

3. A systolic murmur heard best at the 2nd l. interspace is sometimes present in *mitral regurgitation* with a hypertrophied l. auricle. It must not be mistaken for pulmonary stenosis.

4. The "*Milk-spot*" murmur is due to a localised thickening of the visceral pericardium, appearing as a glistening white spot near the centre of the anterior surface of the heart. Usually it is unattended by symptoms, but it may be of importance clinically, for it is apt to be mistaken for valvular disease. The "*milk-spot murmur*" (based on 23 observations, verified by autopsy at the Paddington Infirmary), is generally a prolonged rough bruit, systolic in rhythm, though occasionally double; it is *strictly localised* to a circle of 1" or 1½" radius, whose centre is situated in the third l. interspace, close to the sternum, which is also its position of maximum intensity. Another important

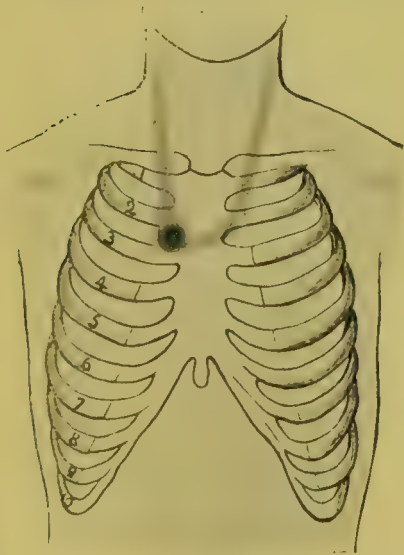


Fig. 17.—The systolic murmur of aortic stenosis. Depth of shading indicates intensity of murmur.

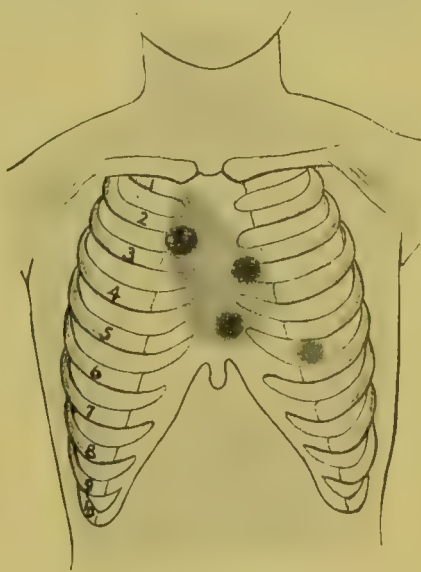


Fig. 18.—The diastolic murmur of aortic regurgitation.. Depth of shading indicates intensity of murmur.

feature is that *at one time it is very rough and loud*, and a day or so later it may have completely disappeared. These features and the absence of the concomitant symptoms of C. V. D., or of chlorosis, enable us to differentiate the milk-spot murmur from other conditions. It was found more often in hypertrophied hearts than in those of normal size. It has been variously attributed to tight-lacing, the soldier's shoulder straps, and other less probable causes. The condition is more frequently met with in adult or advanced life. A history of pericarditis was obtainable in only 1 of the 23 cases. The reader is also referred to an article by Prof. Gairdner, in the *Edinburgh Med. Jour.*, vol. iv., part 2, pp. 909, *et seq.*, and *Monthly Jour. Med. Sci.*, vol. xii. (1851), p. 103.

5. A *congenital* murmur, usually systolic and localised to the base, has been known—in rare cases—to persist throughout life in some persons who have never experienced any other manifestation of cardiac disease, although they have lived to a good old age. The nature of the lesion in such cases is obscure.

6. *Cardio-pulmonary* or *Cardio-respiratory* murmurs are also rare, and are probably produced by the expulsion of air from the adjacent lung tissue by the movements of the heart. They do not indicate any cardiac lesion, and the lung may also be healthy, but they are sometimes associated with phthisis when the cavity is near the heart. They are heard in various parts of the antero-lateral region of the chest. They have a

blowing, whiffing, or "sipping" character; are usually systolic in rhythm, and in rare cases double, though the systolic element is always loudest. Sometimes these murmurs disappear when the patient alters his position or posture; when he stops breathing they may be weakened, abolished, or unaltered.

**B. Murmurs** heard in the **diastolic interval** may occupy either (*a*) the first half of that interval, replacing, accompanying, or following the second heart-sound (*Diastolic* murmurs); or (*β*) they may occupy the second half of the interval, preceding and leading up to the first heart-sound (*Presystolic* murmurs). (See Fig. 10.)

Murmurs of the *first* kind are produced, in order of frequency, by Aortic Regurgitation, Aneurysm, or Pulmonary Regurgitation; murmurs of the *second* kind are mostly due to Mitral Stenosis, very rarely to Tricuspid Stenosis.

**I. In Aortic Regurgitation** the murmur is *diastolic* (V. D.),<sup>1</sup> and is (i.) audible in the aortic area (2nd r. space), but it is often loudest at the 3rd l. intercostal space; (ii.) it is propagated down the sternum, and audible at the apex (Fig. 18). It is, therefore, one of the loudest and most widespread of murmurs. (iii.) Owing to the amount of dilatation and hypertrophy of the l. ventricle the apex is displaced downwards and outwards more than in any other form of C. V. D. (iv.) The pulse is the characteristic "water-hammer" (§ 65). The face is pale, and the carotids visibly pulsate. Capillary pulsation is generally present, and is detected by drawing a line across the forehead or by lightly pressing on the fingernail or on the lips with a glass slide: the alternate blush and pallor due to the pulsation in the capillaries is thus well brought out. So great may this be that a pulse is sometimes communicated to the veins on the dorsum of the hand.

*General symptoms* in aortic regurgitation. (i.) Pain about the chest, often of an anginoid character, may be complained of before compensation fails; or true angina may be present. (ii.) The anæmia is greater in this than in aortic stenosis. Faintness, giddiness, frontal headache, and disturbed sleep are common. (iii.) Dropsy is rare, as death usually occurs before the mitral valve yields sufficiently to produce the necessary backward pressure. Embolism sometimes occurs, though not so often as in mitral stenosis.

<sup>1</sup> *Diastolic* murmurs are sometimes spoken of as V. D. murmurs, being produced during the ventricular diastole. Similarly, *presystolic* murmurs are spoken of as A. S. murmurs, being produced during the auricular systole.

II. In **Mitral Stenosis** the murmur is *presystolic* in rhythm. It occurs during the contraction of the auricle, and is known therefore as the auricular systolic or A. S. murmur. It is heard (i.) at the apex; and (ii.) over a very limited area. (iii.) The murmur is rough or rumbling, and runs up to a loudly accentuated first sound. (iv.) A reduplicated second sound is heard best just to the right of the apex.<sup>1</sup> (v.) On palpation a characteristic sign in this heart lesion is the presystolic *thrill* which can be felt. This may be *felt* even before the murmur can be *heard*—i.e., before the number of vibrations per second are sufficient to produce a musical note. In the later stages it disappears altogether. (vi.) The apex beat is not displaced to the l., *unless Regurgitation be also present*; but a little later on, the præcordial area of dulness is increased to the r., owing to the hypertrophy and dilatation of the r. ventricle. (vii.) The pulse is small and hard, until compensation fails.<sup>2</sup>

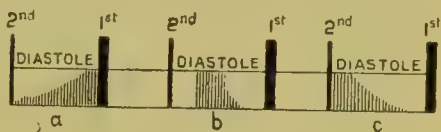


Fig. 19.—Three murmurs may be met with in MITRAL STENOSIS, which, as Dr. Brislowe has pointed out, may occupy different parts of the diastolic interval, and may therefore be called—the (a) LATE-, (b) MID-, and (c) EARLY-DIASTOLIC MURMURS. The late-diastolic (i.e., presystolic) murmur is the commonest; the early-diastolic is least frequent. The reduplication of the second sound has been omitted for the sake of clearness.

In the later stages of mitral stenosis the presystolic murmur disappears and sometimes a mid-diastolic or an early diastolic murmur is heard (Fig. 19). Sometimes the second sound is absent at the apex. Later still, there may be *no murmur at all*; and then the characteristic “slapping” (short, sharp) first sound, and irregular rhythm of the heart, form the sole auscultatory signs. This form of C. V. D. is commoner in women; and in my experience in children.

*General Symptoms.* (i.) Pulmonary congestion (p. 94, *ante*) is especially common; consequently hæmoptysis is more frequent in this than in any other form of C. V. D.; (ii.) emboli are also most

<sup>1</sup> This so-called “reduplicated 2nd” is supposed to be due to a short diastolic murmur following the 2nd sound, because it is heard at the apex, where the pulmonary second is said not to be audible; and because, if due to asynchronous closure of the aortic and pulmonary valves, from the high pressure in the pulmonary circulation, one would expect it to be heard in mitral regurgitation, and in the later stages of mitral stenosis—in both of which it is absent.

<sup>2</sup> Some (e.g., Bradbent) say the pulse is regular in Mitral Stenosis, and that any irregularity infallibly indicates concurrent regurgitation. Others (e.g., Guttman) hold that of all lesions, mitral stenosis alone can give rise to irregularity while compensation is good.



frequent here; (iii.) dropsy is rare until the end; but, on the failure of the r. ventricle, all the symptoms of r. ventricular dilatation appear. Liver enlargement is more common, but cyanosis and dropsy are less common in mitral stenosis than in regurgitation.

III. In **Aortic Aneurysm** a *diastolic* murmur is the most common murmur met with; but reinforcement or emphasis of the 2nd sound in the aortic area is the most constant and invariable sign in this condition. It is also present in the dilated aorta of the aged.

IV. **TRICUSPID STENOSIS** is a rare condition, but it is occasionally met with in young women, and is recognised by (i.) a presystolic murmur, heard loudest over the fifth r. costal cartilage, close to the sternum. (ii.) Dropsy precedes the pulmonary engorgement, but in other respects the consequences are the same as those of regurgitation through this orifice.

V. **REGURGITATION** through the **PULMONARY ARTERY** is practically never met with, excepting either as an accompaniment of congenital malformation of the heart, or as part of a general valvular inflammation in acute ulcerative endocarditis.

**FALLACIES IN THE DIAGNOSIS OF DIASTOLIC MURMURS.** 1. A diastolic murmur due to *aortic regurgitation* may be heard at the *apex*. It must not be mistaken for mitral stenosis. In addition to the fact that the aortic murmur is heard louder at the base than at the apex, it has a uniform character, whereas a mitral diastolic murmur is broken, of varying intensity, and the pulse and other symptoms are different.

2. A *presystolic apical murmur* is occasionally heard with aortic regurgitation (Austin Flint murmur). It is diagnosed from that due to mitral stenosis, by its not being followed by an accentuated 1st sound; and other signs.

3. *Mitral Stenosis* is the most difficult C. V. D. to detect, especially in the 2nd or 3rd stages, when the characteristic murmur may be *altogether absent*. It may then be strongly suspected when there is—(i.) a loud, clear, sharp 1st sound at the apex, with marked accentuation of the pulmonic 2nd sound; or, (ii.) hypertrophy of the r. ventricle, chronic pulmonary catarrh and hæmoptysis, especially if the 2nd sound is reduplicated at the apex.

**C. Double murmurs** may be produced by a combination of any of the above systolic and diastolic murmurs.

a. Double murmurs most audible at the **base** (other than hæmic):

I. **COMBINED AORTIC OBSTRUCTION AND REGURGITATION** is the most common condition, and causes a loud double see-saw murmur, heard best in the second r. interspace.

II. **ANEURYSM OF THE AORTA**, even in the absence of disease of the aortic valves, may be attended by a double murmur having the same characters. This is heard loudest in the second r. interspace, but it may also be heard at the back, to the left of the 4th dorsal vertebra.

III. A double murmur occasionally occurs in the DILATED AORTA of the aged, but with less marked features.

IV. A double murmur, loudest in the pulmonary area, usually indicates CONGENITAL C. V. D.

b. A double murmur most audible at the **apex** may be heard when both MITRAL REGURGITATION and STENOSIS are present. It consists of a systolic bruit followed by a long diastolic murmur almost filling up the diastole.

FALLACIES IN THE DIAGNOSIS OF DOUBLE MURMURS. 1. When a double murmur can be heard both *at base and apex*, do not imagine that mitral regurgitation exists as well as aortic disease. Remember that a systolic mitral and a systolic aortic may be alike in character, and that aortic murmurs can often be heard at the apex as well as the base. To arrive at a conclusion is often very difficult, but one must rely on the position in which the murmur is loudest and on the other features which distinguish mitral and aortic lesions.

2. When a *double aortic murmur* is present, the lesion may be regurgitation, or stenosis, or both together. A diagnosis is made by examining the pulse (p. 140); the rhythm of the thrill, if one is present; and the position of the apex beat. In regurgitation the apex is displaced further downwards and outwards than in any other form of C. V. D. In aortic stenosis the l. ventricular wall is hypertrophied with but little enlargement of the cavity, and as emphysema is so often associated with it, the apex may be hard to find.

3. Murmurs of *pericardial friction* may be mistaken for a double aortic murmur. 4. *Hæmic, cardio-pulmonary*, and *milk-spot* murmurs are occasionally double.

§ 51. GENERAL SYMPTOMS OF CARDIAC VALVULAR DISEASE. The first effect of C. V. D. is *hypertrophy* of the heart, as already mentioned, and so long as there is adequate compensatory hypertrophy there may be no concomitant symptoms at all.

But, sooner or later, in most cases, hypertrophy gives way to *dilatation*, and then a series of characteristic symptoms ensue. Those special to each form of valvular lesion have been referred to in the preceding section. It now remains to mention certain *general symptoms common to all forms of C. V. D.*

1. *Breathlessness* in walking uphill, or even on very slight exertion, is a very constant feature. No serious enfeeblement of the heart-wall or disturbance of its function can exist without this symptom; and it cannot be too much insisted on that breathlessness is not only a symptom, but in general terms is a measure of the extent of the cardiac failure.

2. *Dropsy* occurs early in mitral, late in aortic disease.

3. *Palpitation* is of less diagnostic import, for it may occur

without any organic heart change, and it is not always present with valvular disease.

4. *Pain* is by no means always present in cardiac dilatation, but few cases run their entire course without considerable præcordial discomfort. Pain is a fairly constant feature of aortic disease, and sometimes amounts to angina.

5. *Insomnia*, in advanced cases, is frequently a very troublesome symptom. Sometimes the patient, when dropping off to sleep, suddenly starts with the terror of suffocation, and gasps for breath. It is probably due to the disturbed and irregular circulation through the brain and medulla. *Headache and delirium* are also met with in advanced cardiac disease. The former is occasionally due to temporary high tension; but both are more often due either to pyrexia or to a toxic condition of the blood from failure of the emunctories. In either case free purgation is indicated.

6. *Embolism* may occur, having all the features described under Acute Endocarditis (§ 42). It is most frequent in Mitral Stenosis, and next in aortic disease.

§ 52. The chief **cause** OF C. V. D., in *youth*, is the acute endocarditis which has a special tendency to attack the *mitral* valve, and in *advancing years* the chronic degenerative changes which attack the *aortic* orifice.

1. *Acute Endocarditis* of rheumatic origin is by far the most frequent cause, and a large majority of "heart cases" date their symptoms from an attack of that disease in youth or early adult life. Scarlatina and the other acute specific fevers and all causes of acute endocarditis (§ 42) play their part, but the other specific fevers are infrequent relatively to acute rheumatism and scarlatina.

2. Chronic Endocarditis may come on insidiously, especially under the influence of *certain poisons*, chief among which are alcohol, syphilis, and gout, and especially if these be combined with hard labour. In these circumstances the lesion usually affects the aortic orifice. But chronic endocarditis more often supervenes upon acute endocarditis—an attack of which may have been overlooked.

Dr. C. O. Hawthorne<sup>1</sup> has done good service in drawing attention to the fact that endocarditis may start with an apparently trivial attack of

<sup>1</sup> *Lancet*, 1900, vol. i., p. 1169

subacute rheumatism, the child complaining of nothing but slight pains in the limbs, accompanied perhaps with a slight sore throat, not of sufficient gravity for him to be kept in bed. Parents of children whose antecedents are rheumatic should be warned not to treat such symptoms lightly.

3. *Degenerative changes* (*e.g.*, atheroma) are the lesions mostly met with after middle life. They chiefly affect the aortic orifice, either by injuring the valves or by causing dilatation of the aorta, which, extending to the situation of the valves, prevents them from meeting during the diastole.

4. Any prolonged *high tension*—*e.g.*, that which accompanies arterial sclerosis—may lead to valvular strain, usually aortic. Persistent obstruction in the lungs (*e.g.*, chronic bronchitis), or in the general systemic circulation, may have the same effect as persistent high tension, on the r. or the l. side of the heart respectively.

5. Extensive or prolonged *muscular exertion* may, it is believed, lead to valvular mischief. At least there is no other mode of explaining the fact that a large number of athletes have sclerosis of the aortic valves. In rare circumstances a sudden strain may lead to rupture of a valve.

6. *Congenital* conditions are referred to § 49.

§ 53. The **Prognosis** of chronic heart disease is but ill-understood if the conclusions are based only on hospital cases. They need to be followed from beginning to end as in private practice or infirmary work. It is quite certain that many patients have disease of the heart for years without knowing it. It is also certain that the first symptoms very often date from the patient knowing that they have cardiac disease; and unless there are special reasons to the contrary, a patient should never be informed of its presence.

Cardiac disease may terminate life in three ways: (i.) by sudden death—this may result either from syncope, or from rupture of the heart, or, as some say, from cardiac anæmia, due to non-filling of the coronary arteries, (ii.) by the occurrence of complications, especially bronchitis and other pulmonary affections, or (iii.) by asphyxia, from dropsy of the pleura, often combined with congestion of the lungs.

The probable course and duration depend upon many considerations, but on nothing more than the condition of the *cardiac wall* (No. 3 below), and this should be the object of the most thorough investigation.

1. The presence of certain *'cardiac symptoms'* are in themselves indications that compensatory hypertrophy is giving place to dilatation—*e.g.*, palpitation, dyspnoea, increased by emotion or exertion, cardiac pain, syncopal and anginoid attacks. In actual practice, the prognosis is good in proportion to the *amount of*



*exercise a patient can take without producing breathlessness.* Syncope and anginoid attacks usually indicate serious cardiac embarrassment. Palpitation and cardiac pain are less serious indications; "Delirium cordis" and Cheyne-Stokes breathing are very grave.

2. The *condition of the pulse* is of considerable value in prognosis, but it has to be judged in connection with the valvular lesion. (i.) Irregularity is a grave indication excepting in mitral regurgitation. In aortic disease it is very serious; and at all times intermittency or dropped beats is a symptom of most serious import. (ii.) Persistent rapidity is always a bad sign.

3. The *Physical Signs of Cardiac Hypertrophy and Dilatation* given in Table IV., p. 87, will help us to gauge the amount of dilatation, *i.e.*, failure, or hypertrophy, *i.e.*, compensation, which is present, by means of a careful examination of the apex-beat, by percussion, and auscultation. If emphysema or other condition prevents us obtaining reliable conclusions, it is worth remembering that *a regularly acting heart with an apex in the normal situation*, justifies (with possible exceptions in mitral stenosis) a fairly good prognosis.

For purposes of prognosis—and indeed for treatment also—cases of Cardiac Valvular Disease are best divided into 3 groups or stages. In the *first* stage there is *efficient hypertrophy*, with or without compensatory dilatation (in regurgitant lesions dilatation is also a compensatory process and aids the heart). In this stage the patient may not come under notice at all; both the valvular mischief and the hypertrophy may be discovered accidentally.

In the *second* stage the *dilatation exceeds* that which is necessary, and some of the symptoms referable to the heart, above mentioned, are sure to be present. The dilatation, however, is not sufficient to produce backward pressure.

In the *third* stage there is *advanced dilatation* with thinning of the wall, and backward pressure takes effect in the lungs (in aortic lesions), in the general venous system (in mitral lesions); and finally in both.

4. The presence of *signs of venous obstruction* as a measure of backward pressure—*viz.*, pulmonary congestion, dropsy, lividity of the lips and fingers, enlargement of the liver and spleen, and albuminuria—is unfavourable. But the gravity is very different

in mitral and aortic lesions respectively. In *mitral* cases, a moderate degree of these symptoms indicates only moderate cardiac failure, and it by no means follows that the heart is beyond redemption. But if they occur in *aortic* disease they show that the final stage is reached, and that the patient will probably not live many months. When general venous congestion exists the relative *amount of urine* passed day by day is a good measure of the strength of the heart and the improvement made—a fact which is not generally mentioned.

5. Concerning the *nature of the valvular lesion* as bearing on the prognosis, some difference of opinion is expressed as to the relative importance of aortic and mitral lesions.<sup>1</sup> My own experience is that a moderate degree of aortic stenosis is the most favourable form, and if well compensated may give rise to little or no inconvenience; the patient mostly dying of some intercurrent malady. Next in order comes mitral regurgitation, then mitral stenosis; the most serious being aortic regurgitation, the valvular disease that most frequently ends in sudden death. Combined lesions of stenosis and regurgitation are naturally more serious than single ones, and the gravest of all valvular lesions is double aortic disease.

In *Aortic Regurgitation*, the *measure of the amount* of regurgitation, and therefore the prognosis depends upon the clearness with which one can hear the aortic second sound (as distinct from the murmur) in the carotid arteries. In *Mitral Regurgitation* a loud murmur *following* the first sound is more favourable than a weak murmur, or than one which replaces or accompanies the first sound. In *Mitral Stenosis* a faint or absent second sound is a grave sign. Apical murmurs due to *dilatation* can generally be made to disappear under treatment.

In *Double Aortic Disease* it is important to note which is the louder, the first or the second of the two bruits. If the first be the louder, it indicates considerable compensating hypertrophy of the left ventricle, and the prognosis is more favourable; but if the second, the regurgitant, bruit, be the louder of the two, it

<sup>1</sup> Peacock and Bristowe place *aortic obstruction* as the most favourable, *mitral stenosis* next, *mitral regurgitation* next, and *aortic regurgitation*, as by far the most fatal. Sir Dominic Corrigan, on the other hand, and some other observers, regarded this lesion as consistent with unlimited longevity; but Dr. George Balfour states that cases of aortic incompetence generally "die within 4 years of admission to hospital." As regards this last statement, it depends really on the age at which it supervenes; see p. 106, par. 7.

probably indicates a weakened ventricle, which allows a large reflux of blood, and the prognosis is as grave as well can be.

6. The *Primary Cause* of the valvular mischief influences the prognosis to some extent. Injury and congenital mischief, both happily rare, are very serious. Rheumatism is grave in proportion to its tendency to recur. In general terms cases due to acute endocarditis in early life are much more favourable than the degenerative changes (accompanied perhaps by an alcoholic or syphilitic taint) supervening during middle life.

7. *Age* is not a very important factor. Valvular lesions in childhood are more readily compensated, but at the same time advance more rapidly. *Mitral stenosis* coming on in childhood is much graver than when it supervenes in the adult, and generally terminates fatally before the age of 21. On the other hand, *aortic regurgitation* due to endocarditis in youth is compatible with a long and useful life; but when coming on in middle or advanced life it is generally due to degeneration and dilatation of the aortic orifice, a condition of far graver import.

8. The *temperament, habits, and means* of the patient will naturally influence his future. The prognosis is bad in the intemperate, and those who lead irregular lives. It is also unfavourable in the destitute, and in those who are compelled to work hard for their daily bread. Nevertheless, complete idleness is equally bad, and a patient should be encouraged to do as much as he is able without fatigue.

9. Finally, before hazarding a prognosis in any given case, the *effects of treatment* should always be watched, for it is sometimes truly wonderful how the skilful administration of digitalis and the application of modern methods of treatment, will sometimes seem to snatch the patient from the very jaws of death. The existence of an organic murmur without change, and not requiring active treatment for 2 years, justifies a favourable prognosis.

§ 54. THE TREATMENT OF CHRONIC HEART DISEASE (including Cardiac Dilatation or Failure, and C. V. D.) should be directed to two objects; one is to strengthen and correct the perverted action of the heart, the other to relieve and counterbalance the obstructed circulation. These may be accomplished by four means:—

1. *Dietetic and hygienic measures.* Rest in bed is always indicated in *severe* cases. The diet, which should consist of easily assimilated material, should contain only a moderate amount of fluid. The chief meal should be taken in the middle of the day. Anything requiring prolonged digestion disturbs the night's rest, and it is sometimes a good rule to allow nothing solid after 2 P.M. Alcohol, tobacco, strong tea and coffee, should, as a rule, be avoided. A small quantity of stimulant with meals may be called for, but its use is best avoided if possible, on account of the reaction afterwards, and because of the marked *tendency to excess*, which undoubtedly exists in cardiac

cases. As regards exercise, since the voluntary muscles play such an important part in promoting the circulation of the blood and so aiding the heart, moderate exercise should be favoured in all but advanced cases. The best rule is to allow the patient to do just as much as he is able without tiring himself or producing palpitation and dyspnœa. A healthy out-door life certainly promotes the nutrition of the heart and the body generally.

2. *Drugs.* In cardiac failure, especially when the pulse becomes feeble, rapid, and irregular, digitalis is *par excellence* the remedy. It is contra-indicated when there is full compensatory hypertrophy, and the pulse is fairly strong and regular. It is best to begin with 5 or 10 M doses of the tinct., twice or thrice daily, and subsequently to reduce it. It may be continued for a considerable time in smaller doses in the form of a tonic. Its action may be promoted by the addition of spt. am. aromat. and senega. Digitalis not only affects the heart, but relieves the engorgement of the viscera. Strophanthus has much the same action as digitalis on the heart; it acts more rapidly, and does not constrict the arteries. F. 54, 57, 59, 67, and 84 are useful.

In *aortic valvular disease* and in *mitral stenosis*, where the pulse has other characters, digitalis is not so valuable a drug.<sup>1</sup> But in the later stages even of these affections, when compensation begins to fail, small doses of digitalis give relief. Belladonna is sometimes useful if there is relaxation of the vessels. Among the cardiac tonics strychnine, nux vomica, iron, and arsenic, are the most valuable, in the order mentioned.

The various symptoms may be met by appropriate remedies. For the pulmonary congestion, squills and stimulating expectorants are indicated. In all cases of intense *venous engorgement*, whether of the general or pulmonary systems, and especially if it has come on rapidly, *venesection* affords very prompt relief; and as much as 2 to 10 oz. may be removed. The indications for venesection are orthopnœa, lividity of the face, and distended jugulars; its efficacy is well seen in Fig. 20, p. 108.

For *breathlessness* spirits of ether or of chloroform, and ammonia, are useful. Nitroglycerine is useful where breathlessness is

<sup>1</sup> In mitral stenosis especially, digitalis may cause irregularity, when strophanthus must be substituted.



associated with the high tension which may accompany cardiac hypertrophy: and at the same time it cures the headache and sleeplessness due to the same cause. Ether  $\mathfrak{M}$  xx.—xl., or strychn. sulph. gr.  $\frac{1}{60}$ th hypodermically, are useful for the paroxysms of dyspnœa. Cough is relieved by drinks of hot milk, and drugs, such as codeia, small doses of opium, and chloroform or ether.

For the *dropsy*, drugs are employed which strengthen the heart, together with those that act upon the kidneys and promote diuresis. Digitalis, strophanthus, and caffein act in both ways. Diuretin and calomel, combined with digitalis<sup>1</sup> and a milk diet, are valuable remedies. Hydragogue cathartics, such as pulv. jalapæ co., gr. xx., and cream of tartar assist the action of the diuretics.

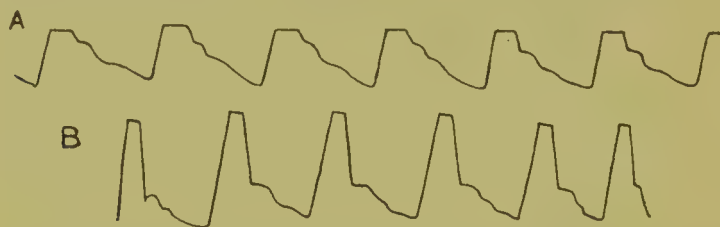


Fig. 20.—Tracings A and B show the efficiency of **bleeding**. A shows the flat top of high tension. B was taken immediately after 5 oz. blood were removed, and shows the reduction thus effected, and also the senile character of virtual tension. The patient was about 57 years of age, and suffered from C. V. D. with recurrent high tension (with headache, etc.). The urine was always normal. Some years later he was brought in with apoplexy and died.

Diaphoretics are not of much use in cardiac dropsy. Should such treatment fail, puncturing the legs (§ 22) and paracentesis abdominis must be considered. F. 55 is useful.

For *palpitation* alcohol is a most valuable cardiac stimulant, and relieves the breathlessness as well; this, patients unfortunately soon find out for themselves, and thus C. V. D. is a not infrequent cause of chronic alcoholism, especially among women, who take it secretly, during the night, when the palpitation is most apt to come on. The exact dose should therefore be carefully prescribed, and the quantity always moderate. Other causes of palpitation which may be present should be

<sup>1</sup> Dr. Alex. Morison (*Lancet*, 1899, vol. ii., p. 1147, and 1900, vol. i., p. 1882), on "Value of Mercury in Heart Disease," publishes cases and quotes Broadbent and others to prove that when Digitalis given alone may be useless in cases of heart disease with great dropsy, yet if given together with minute and frequently repeated doses of Calomel, the result is excellent. Thus gr.  $\frac{1}{4}$  Calomel with gr.  $\frac{1}{2}$  Digitalis leaves every 2 hours, rapidly causes diuresis.

treated (§ 23). For *sleeplessness*, opium or morphia hypodermically is useful; in mitral disease, however, where the liver is congested, opium is better avoided, and other drugs employed, such as pot. brom., sulphonal, trional, and paraldehyde. I have not found small doses of chloral do harm as some maintain. The *hæmoptysis* of heart disease is best left alone, as it relieves the congestion. The *gastric* symptoms may be relieved by acting on the congested liver with calomel, gr.  $\frac{1}{2}$ —1 every night, with sod. sulph. and sod. bicarb., gr. xxx. in 2 oz. hot water in the mornings. Digitalis must be stopped if it causes sickness. It may be necessary to give predigested food. For the treatment of *syncope* attacks and *pain*, vide § 25 & § 24; F. 56 is useful.

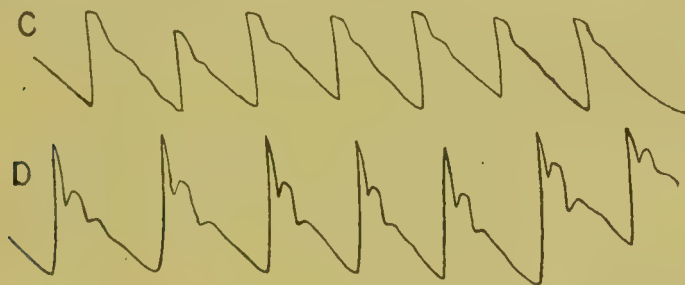


Fig. 21.—Tracings C and D, taken by a Marey's sphygmograph (in which the momentum of the lever is greater, and the excursion larger, than those taken by Dudgeon's). C (which shows simply high tension) was taken before, and D (which shows the reduction of tension) was taken directly **after massage**—massage and passive movements. The patient was a man *æt.* 65, under care for arterial sclerosis, and these tracings show the efficacy of massage in relieving the heart.

(3) *Massage and systematised exercises.* At one time, rest was regarded as imperative for all forms of cardiac disease. But the advance of physiological knowledge has shown what an important part the skeletal muscles play in the circulation of the blood, by squeezing the fluids out of the soft-walled veins and lymphatics, while they cannot compress the lumen of the firm-walled arteries. There are three varieties of this treatment which are invaluable for different degrees of cardiac failure. *First*, for the worst cases, *gentle massage*, combined perhaps with *passive movements*. These are available where any kind of voluntary movement on the part of the patient is attended with breathlessness. The great value of properly regulated massage in cardiac failure is well seen in the case from which the tracings in Fig. 21 were taken. *Secondly*, slow *voluntary movements* of flexion and extension on the part

of the patient whilst standing or sitting. In the Nauheim system these voluntary movements are gently resisted by the operator—"resistance gymnastics"—see F.113. These movements, combined with *baths* (see below), constitute the essence of the Schott system. *Thirdly*, Oertel's method, which consists of three parts: First, reducing the amount of fluid taken to 31 oz. per diem (to include the amount contained in the solid food) and promoting perspiration; secondly, a diet largely consisting of proteids;<sup>1</sup> and thirdly, graduated exercise in the form of walking uphill, each day a little further. Cases attended by plethora and obesity are the most suitable.

(4) *Baths*, such as those in use at Nauheim, may be usefully added to the preceding. They act by relaxing the arterioles of the skin directly, and the arterioles of other parts reflexly. By these means blood is transferred from the venous to the arterial system, and its flow accelerated.

This treatment is commenced with weak saline baths at a temperature of 92° to 95° F., consisting of 1 lb. common salt, and 1½ oz. Calcium Chloride to every 10 gallons of water. These should be given every other day for a week, the patient remaining in the bath six minutes. The strength is then gradually increased to 3 lb. salt and 4½ oz. Calcium Chloride for every 10 gallons of water; and the patient remains in the bath for twenty minutes, with the temperature lowered to 85° F. if he can bear it. In a fortnight or more effervescing baths are employed. In every 10 gallons of water dissolve 2 oz. Sod. Bicarb; and add 3 oz. Hydrochloric acid just before the patient enters. Gradually increase the strength to 8 oz. Sod. Bicarb and 12 oz. Ac. Hydrochlor.

It is simpler to employ "Sandow's Tablets" and powders, which contain the ingredients for the baths specially prepared in a convenient form for ready use.

Treatment extends over five weeks. The effervescing baths are ordered according to the discretion of the physician; and in severe cases it is sometimes unsafe to employ them at all.

VI. *On auscultation, NO MURMUR can be heard, and the heart sounds are very feeble. The IMPULSE AT THE APEX IS SO WEAK that it cannot be localised. FATTY DEGENERATION of the heart-wall may be strongly suspected.*

§ 55. **Fatty Heart** in its clinical sense indicates enfeeblement of the cardiac wall. A better term for it would be **CARDIAC ENFEEBLEMENT**, or **Primary Cardiac Failure**.<sup>2</sup> Examples have now

<sup>1</sup> Oertel's dietary is as follows: *Morning*: 6 oz. coffee, 3 oz. bread.—*Noon*: 3–4 oz. soup, 7–8 oz. roast meat or poultry, salad or green vegetable, a little fish, 1 oz. bread or farinaceous pudding, 3–6 oz. fruit, no liquid (excepting in hot weather, 6 oz. light wine). —*Afternoon*: 6 oz. tea or coffee (1 oz. bread occasionally). *Evening*: 1 or 2 lightly boiled eggs, 1 oz. bread, salad, fruit, sometimes a small piece of cheese, 6–8 oz. light wine with 4–5 oz. water.

<sup>2</sup> Fatty heart may occur anatomically in three forms, which are here mentioned in order of frequency, though they may occur together. 1. *Fatty deposit* occurs very generally in very fat persons (or adiposum of older authors), and consists of a deposit of fat confined, usually, to the external surface of the organ. It is not generally of much consequence. 2. *Fatty infiltration*, where the fat is deposited in the interstices of the fibres. This also may be part of general obesity, or, more frequently, the result of alcohol excesses, and it impedes the action of the heart. 3. *True fatty degeneration* is least common. In this the

been given of all the various physical signs liable to be found in the heart by means of inspection, palpation, percussion, and auscultation. But, supposing the most careful examination reveals *no physical signs*, although, by reason of certain *subjective symptoms*, we believe the patient to be suffering from cardiac disease, DEGENERATION OF THE CARDIAC WALL should be borne in mind, the diagnosis of which often rests, as I have previously mentioned, on a process of exclusion.

Its detection is often a matter of some difficulty, but the disease may be *suspected* (i.) when the pulse and heart impulse are feeble, and the heart sounds perhaps inaudible; (ii.) if the patient be subject to attacks of fainting or of dizziness; and (iii.) if he be subject to palpitation and breathlessness. The pulse may be either very quick or very slow, intermittent or irregular. The disease is more frequently met with in persons past middle age, and if unaccountable fainting attacks occur for the first time at this age period, fatty heart is the most probable cause. Some œdema of the ankles *may* also be present. (iv.) The heart sounds are not usually accompanied by a murmur, for even if valvular mischief exist, the force of the heart may not be sufficient to produce a bruit. The area of præcordial dulness may or may not be increased; but in any case it is often obscured by emphysema of the lungs, which is itself one of the causes of fatty degeneration. Later on, anginoid and epileptiform attacks are not uncommon.

The *prognosis* is extremely grave. The earlier stages of the malady are so insidious, that by the time pronounced symptoms

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heart muscle becomes pale, very friable, and *under the microscope*, its fibres are seen to be the seat of a granular fatty change, staining black with osmic acid. Two forms are described of this latter: (a) A diffuse form, which may be the result of alcoholism, emphysema, pernicious anaemia, typhus, and other acute specific fevers, or general faulty nutrition. (b) In a localised form it occurs either just beneath the internal or external surface as the result of endocarditis and pericarditis, or as localised patches where a branch of the coronary artery is blocked or diseased. It is thus that *aneurysm of the heart* or *rupture* occur. Alcoholic excess is certainly the most common and most potent cause of both fatty infiltration and degeneration. It is certain to follow sooner or later if alcoholic excess be persisted in, and is generally the immediate cause of death of the "tippler." Out of 5 rabbits fed for varying periods of time on ethylic alcohol, 4 died of fatty heart. Dr. H. J. Berkley, "Brain," 1895, p. 477. Dr. F. J. Poynton gives illustrations of microscopic specimens showing the degenerated condition of the heart muscle in cases of diphtheria, chorea, and rheumatic fever. *Lancet*, vol. i. (1900), p. 1352.

Other affections of the myocardium Parenchymatous degeneration, Interstitial myocarditis, Brown Induration, Fibroid myocarditis, and those rarer forms, Anæmic necrosis, amyloid and calcareous degeneration may produce the same symptoms as fatty heart, and their existence may similarly remain unrevealed unless dilatation be present, until perhaps sudden death occurs.



appear, great and irreparable mischief may be done. The patient may die in one of the syncopal attacks, or if not he will rarely live for more than 6—12 months after definite symptoms have become manifest.

*Diagnosis.* In the early stages it may be impossible to distinguish Fatty Heart from Cardiac Dilatation, especially when emphysema is present, preventing accurate percussion. In *dilatation* (i.) the dyspnoea has often the peculiar character of being improved by using the voice (Broadbent). (ii.) The syncopal attacks of dilatation when present are sudden, with complete unconsciousness, and soon recovered from; those of fatty heart are prolonged, but less intense. (iii.) In dilatation there is nearly always backward pressure leading to anasarca, fluid in the serous cavities, and congestion of organs.

*Treatment* consists of (i.) perfect rest, both of body and mind, and avoidance of anything like excitement; (ii.) stimulants in small and frequent doses; ammonia, alcohol, ether, combined with plenty of easily assimilated nourishment, as in the form of mist. vini Gallici; (iii.) cardiac tonics, and especially strychnine, arsenic, and quinine, combined with plenty of fresh air. Digitalis should not be given if there be no signs of dilatation and the pulse be slow. It is, however, of great value in the opposite conditions. (iv.) The heart may be relieved of some of its work by passive movements, massage, and other measures described under C. V. D., due care being exercised.

## CHAPTER IV.

### ANEURYSM OF THE AORTA AND OTHER INTRATHORACIC TUMOURS.

**Anatomy.** The mediastinum is the irregular space in the chest which lies between the two pleural sacs. For anatomical purposes it is divided into four parts, or as they are called, *mediastina*, viz., the *middle mediastinum*, which is occupied by the heart and pericardial sac; the *anterior* is the space in front, the *posterior* is the space behind, and the *superior* is the space above the pericardial sac. The most important structures contained in these spaces are—the thymus or its remains, the arch of the aorta with its branches (innominate, left subclavian and carotids), the superior and inferior venæ caviæ with the innominate and azygos veins, the pulmonary vessels, the trachea and bronchi, the vagus, recurrent laryngeal, phrenic and splanchnic nerves, the cardiac and pulmonary plexuses, the roots of the lungs, the œsophagus, thoracic duct, lymphatic glands and vessels and loose cellular tissue (Fig. 11).

The lymphatic glands are important on account of the occurrence of lympho-sarcoma and other glandular enlargements which may form mediastinal tumours. There are four groups: (i.) anterior mediastinal, (ii.) superior mediastinal, along the aortic arch and innominate vein, (iii.) posterior mediastinal, along the aorta and œsophagus, and (iv.) bronchial, near the main bronchi and the bifurcation of the trachea.

DULNESS WITH AN IRREGULAR OUTLINE is referred to in the *italicised* remarks on diagnosis on p. 90.

If, on percussing over the sternum,<sup>1</sup> or just beside it, the præcordial dulness is found to be **increased irregularly upwards**—the

<sup>1</sup> Remember, in percussing over the sternum the note elicited is of a much lighter pitch than that just beside the sternum.

morbid condition may be PERICARDIAL EFFUSION, HYPERTROPHY OF THE LEFT AURICLE, RETRACTION OF THE LUNG, AN ABDOMINAL SWELLING PUSHING UP THE HEART AS A WHOLE, OR AORTIC ANEURYSM AND SOME OTHER MEDIASTINAL TUMOURS. The last-named are generally to be distinguished sooner or later by the presence of pressure symptoms (p. 117). If possible, a radiograph should be made.<sup>1</sup>

*If, on auscultation over the abnormal dulness near the base of the heart, there is a REINFORCED OR RINGING SECOND HEART SOUND—perhaps a systolic or diastolic murmur—the disease is probably ANEURYSM OF THE AORTA.*

§ 56. **Intrathoracic Aneurysm.** Aneurysm of the aorta is undoubtedly the commonest of intrathoracic tumours. In regard to the anatomy of this serious and important malady the student should study Fig. 11, p. 60.

The arch of the aorta is the favourite seat for aneurysmal dilatation. Its shape and the fact that it is subject to continuous shock make it surprising that the malady is not even more frequent. Any part of it may be affected—the ascending, transverse or descending part of the arch. The dilatation may assume either a fusiform or saccular shape, the former being the more frequent. The dilatation may make its way in various directions, and it is extraordinary how bones, cartilages and other hard structures may become eroded and absorbed under its pressure.

*Symptoms.* One of the earliest results of aneurysm near the root of the aorta is *cardiac hypertrophy*, but this may not occur at all when it involves other parts. A ringing or *accentuated second* sound at the base is a more constant sign of the presence of aneurysm. According to its position, aneurysm of the aorta may be either very easy or very difficult to detect. If it involves the first part of the aorta, near the *front* of the chest, it is soon revealed by definite *physical signs*. If the second or third parts of the arch are involved, and the tumour extends backwards, there may be no physical signs, and even the *pressure symptoms* may be obscure. Thus the clinical manifestations belong to two categories—physical signs and pressure symptoms; and we have

<sup>1</sup> Dr Hugh Walsham, *The Lancet*, Nov. 3, 1900.

two varieties of aneurysm:—(a) The *aneurysm of physical signs*, when the FIRST HALF of the arch is involved; (β) The *aneurysm of symptoms* (that is, pressure symptoms), when the SECOND HALF of the arch is involved.

The *symptoms common* to aortic aneurysm in all positions will be considered first, because these are the symptoms which will probably first attract our notice. Then we will turn to certain others *special* to the 1st, 2nd, and 3rd parts of the arch respectively.

#### Symptoms COMMON TO ALL POSITIONS:—

1. Dyspnoea is often one of the earliest complaints which the patient makes. When it is due to pressure on the trachea, as in aneurysm affecting the transverse portion of the arch, it is persistent and stridulous in character. When it is due to pressure on the anterior pulmonary plexus, as in aneurysm of the first part of the arch, it is often paroxysmal.

2. Cough is generally present, and has a characteristic brassy sound (gander cough). Pressure upon the recurrent laryngeal nerve is common, with consequent paralysis of the left vocal cord, and there may be hoarseness or even aphonia from the same cause. *Paralysis of the left vocal cord*, in the absence of central nerve lesions, practically always means aortic aneurysm.<sup>1</sup>

3. Pain in the chest is another common symptom. It may occur in attacks of an anginoid character, shooting down one or both arms, usually the left, especially in aneurysm of the first part of the arch.<sup>2</sup> The pain may be neuralgic when there is pressure on nerves; or it may be of a dull boring character when due to erosion of bone, such as occurs in connection with aneurysm of the descending arch.<sup>3</sup>

4. A reinforcement of the aortic second sound is the most constant of the auscultatory signs of aortic aneurysm. It is sometimes spoken of as a “ringing second” sound, and is due to the increase in the aortic tension (Broadbent).

<sup>1</sup> Laryngoscopic examination should be a matter of routine in all suspicious cases, because abductor paralysis occurs before complete paralysis, and the former may be unattended by any alteration of voice.

<sup>2</sup> Short of definite anginoid attacks of this kind, patients with aortic aneurysm are liable to feelings of suffocation, constriction, or “spasm” in the chest, and nameless dreads come over them from time to time without cause. Such attacks may in many cases be brought on by bending the head backwards, or by any movement which stretches the neck. I have known patients with dilated and rigid aorta suffer from the same symptoms.

<sup>3</sup> A case is mentioned in the footnote to p. 47 in which this was almost the only symptom



5. The diastolic shock or thud is an equally important sign. It is felt by the hand or the stethoscope, and is synchronous with the second sound.

6. Inequality of the radial pulses is a fairly frequent symptom. It is present whenever the aorta is so placed as to cause a difference in the arterial tension in the great vessels which spring from the aorta. The typical aortic pulse occurs in the one just beyond the aneurysmal sac, and its characteristic is a loss of the pulse wave, the blood flowing in one continuous stream.

7. Inequality of the pupils occurs from pressure on the sympathetic. In the early stage the irritation of the nerve causes dilatation of the pupil on the same side. Later on, there is paralysis with contraction of the pupil; accompanied sometimes by vascular dilatation and unilateral sweating of the face and neck.

8. The heart may be displaced when the aneurysm is large, usually to the left. It is sometimes hypertrophied.

a.—Symptoms peculiar to aneurysm of the **ascending or first part of the arch**. Aneurysm of this part of the arch is usually easy of detection, and in marked cases the *physical signs* are unmistakable. (i.) On auscultation a marked or accentuated second sound is usually to be heard; and in a large number of cases, where the dilatation involves the valvular orifice, a diastolic murmur is also heard. Over the site of the aneurysm a systolic murmur is always present; and this is frequently present also at the aortic area. Thus, a double murmur at the aortic area is found in many cases. (ii.) Any percussion dulness present is continuous with that of the heart. It usually extends to the r. of the sternum, but this depends upon whether the aneurysm makes its way forwards or not. The l. heart gradually hypertrophies. (iii.) On palpation, the diastolic shock is very diagnostic. Sometimes there is a thrill, felt also in the suprasternal notch. (iv.) When the aneurysm is so large as to form a tumour the swelling expands laterally with each systole of the heart. (v.) The right bronchus may be pressed upon, leading to diminished or absent respiratory murmur (R. M.) of the right lung. In severe cases there may be pressure on the superior vena cava, with œdema of the neck and arms. (vi.) The dyspnœa is paroxysmal; and the right recurrent laryngeal nerve may be involved, with right laryngeal paralysis.

b.—The symptoms of aneurysm of the second or **transverse part of the arch** may be equally easy to detect when it makes its way forwards. But when the posterior part is affected it may present considerable difficulty in diagnosis, especially from other intrathoracic tumours. (i.) The dyspnœa may be either paroxysmal, or continuous, with inspiratory stridor, owing to the pressure upon the trachea. (ii.) Pressure upon the left bronchus may lead to diminished R. M. in the left lung, and symptoms (2) and (5) above are specially marked in aneurysm of the transverse arch. (iii.) Tracheal tugging is a very characteristic sign of aneurysm in this situation. Standing behind the patient, hold the cricoid between the finger and thumb and press gently upwards, the patient sitting in a chair erect with the chin up (see Fig. 22). In this way the pulsation is transmitted by the trachea to the hand. (iv.) The physical signs—which are in this situation less marked, or may be absent—consist of a thrill felt on palpating the suprasternal notch, dulness on percussion over the manubrium, continuous with that of the heart, and extending from the middle line to the left of the sternum; and auscultatory signs as above noted, a.—(i.).



Fig. 22.—**Tracheal tugging**, showing position of hands in order to elicit this symptom.

c.—The symptoms of aneurysm affecting the **descending aorta** may be very obscure. (i.) Intense pain in the back is the most common symptom, and there may be no other for a long time (*Case* in footnote, p. 47). The pain may pass to the side, following the course of an intercostal nerve. (ii.) Other pressure symptoms, such as dysphagia, from pressure upon the œsophagus; wasting, from pressure upon the thoracic duct; disease of the left lung, from pressure upon its bronchi; and any of the other symptoms mentioned on p. 115. (iii.) If the swelling attain any size physical

signs to auscultation and percussion may become apparent in the left (occasionally the right) scapular region; and in advanced cases there may even be a pulsating swelling without the knowledge of the patient. Osler found that in such cases there is absence of pulsation in the femoral arteries.<sup>1</sup>

CASES. There were always cases of aneurysm at the Paddington Infirmary, and at one time I had three cases which well contrasted the symptoms of involvement of the different parts, and the events which are liable to ensue.

Thus, in *Case A* (*Aneurysm of the First Part*) there were (i.) well-marked anginoid pains; (ii.) well-marked physical signs; and (iii.) cyanosis from pressure on the vena cava and the r. auricle; terminating in sudden death by the aneurysm bursting into the pericardium. After death, aneurysm of the first part of the arch was found pressing on the pulmonary artery and the right auricle.

In *Case B* there were (i.) abductor paresis of the left vocal cord; (ii.) permanent and well-marked contraction of the pupil on the right side; (iii.) great inequality of the pulses; and (iv.) death from bursting of the aneurysm into the trachea before any physical signs were apparent. Here the aneurysm sprang from the upper and posterior part of the transverse portion, pressed upon the trachea, and indirectly on the sympathetic ganglia.

*Case C* was mistaken at first for asthmatic bronchitis. There were (i.) orthopnoea, paroxysmal dyspnoea, and collapse of the lung from pressure on the left bronchus; (ii.) dysphagia; and (iii.) severe pain in the back: no physical signs at any time. At the P. M. there was found a considerable dilatation of the descending arch pressing on the œsophagus and vagus, and eroding the vertebra. Another case somewhat resembling this is mentioned in a footnote to § 24, which terminated in sudden death by rupture into a bronchus.

These cases present in a very typical manner the 3 clinical and anatomical varieties of the disease. All aneurysms of the arch of the aorta are sooner or later attended by one or other of the above group of pressure symptoms, excepting (i.) such as arise from the upper part of the transverse portion and project forwards and upwards; or (ii.) such as arise from the descending part of the arch beyond the pulmonary plexuses. In these positions there are no important structures upon which pressure could produce symptoms.

*Ætiology.* (1) Aortic aneurysm is far more frequent in men, especially those in the prime of life—namely, between the ages of 35 and 50. (2) It is especially frequent among soldiers and those who do laborious work. The liability of soldiers has been attributed to the wearing of belts and the like, but it is probably due to the fact that these classes are subjected to sudden and severe muscular exertion and heart-strain at certain times. It also occurs among blacksmiths for the same reason.

<sup>1</sup> Osler, "Prin. and Pract. of Med.," p. 710.

(3) Both syphilis and alcohol are potent agencies in the production of arterial degeneration. Alcohol acts probably in two ways—partly by predisposing to degeneration of the aortic walls, and partly by over-stimulating the heart from time to time.

(4) As an exciting cause some cases of aneurysm date from a period of over-exertion, exposure, and destitution.

*Diagnosis.* The diagnosis of a deep-seated aneurysm is sometimes as difficult in the early stages, as it is easy when the aneurysm is situated superficially. The diagnosis from *Cardiac Valvular Disease* and other causes of cardiac hypertrophy (§ 46) is made by the occurrence of the pressure symptoms. Many of the local signs of aortic aneurysm may be produced by a *dilated and rigid aorta*, but here again the pressure symptoms are wanting. The *throbbing aorta* of aortic regurgitation is apt to be mistaken for aortic aneurysm, and it is sometimes impossible to differentiate these conditions. The throbbing aorta in Graves' Disease, and severe cases of Anæmia may also give rise to difficulty. *Mediastinal growths*, on the other hand, may have the same pressure symptoms as aneurysm, and can only be diagnosed by the absence of the physical signs referable to the heart. There is no murmur on auscultation over the dull region, and the area of dulness is usually not so limited or defined. Finally, the course of mediastinal tumours rarely lasts longer than 18 months. Of late, radiography has been employed in the diagnosis of the presence and nature of intra-thoracic tumours.<sup>1</sup>

*Prognosis.* By treatment much can be done to prolong life, and the patient may live a good many years if his occupation does not necessitate much exertion. Death may occur in four ways—from rupture, exhaustion, cardiac failure, or complications. The rupture usually leads to a sudden and copious hæmorrhage which terminates life; but sometimes there is a slight leakage, which may recur at intervals of a few days. With aneurysm of the *ascending aorta* rupture usually takes place into the pericardium, pulmonary artery, or superior vena cava; with aneurysm of the *transverse arch*, into the trachea (a very frequent situation) or into the bronchi; and, when the *descending aorta* is involved, the

<sup>1</sup> Diagnosis of Aneurysm by Skiagrams. Dalgarno and Galloway, *Lancet*, 1897, vol. i., p. 1268; and Walsham, *ib.*, Nov. 3, 1900.

blood usually finds its way into the pleura or œsophagus. The process may be so gradual that there is no sudden onset of symptoms, such as dyspnœa or cyanosis, or bleeding, and death may not take place for some time. But generally, as in the cases just narrated, it is copious and sudden, death speedily ensuing.

The severity of any case is measured to some extent by the amount of dyspnœa present and the rapidity of the evolution of symptoms.

Other consequences or complications are due in most part to the effects of pressure—such as collapse or a low form of pneumonia of the lung, hydrothorax, œdema of the head and neck, etc.

*Treatment.* The indications are three in number: (a) to lessen arterial tension; (b) to slow and steady the heart; and (c) to increase the blood coagulability in the hope that laminated clot will form in the sac. Absolute rest in bed must be enjoined. This alone may accomplish very considerable relief, and there is no doubt that some of the extraordinary results claimed for certain remedies have been due to rest. Much can be accomplished by diet. It should be of the smallest quantity consistent with life. Tufnell's dietary<sup>1</sup> is based on this fact, and in it only 8 ounces of fluid and 10 ounces of solid are allowed per diem. It must be persevered in for three to six months. The good derived from this dietary mainly depends on the reduction of fluid. Drugs should be employed to steady the heart and reduce the tension (see "Pulse"). There is, however, one remedy which is undoubtedly capable of materially improving thoracic aneurysm—viz., iodide of potassium in large doses, commencing with 20 gr. 3 or 4 times a day, and gradually increasing it.

I am not aware if calcium chloride in large doses has ever been tried, but in view of its known power to increase coagulation it seems to me worth a trial. The digestive organs often need attention. For the pain, morphia, atropine, or belladonna internally, or in the form of a plaster are used; if of anginoid character, nitroglycerine. Even if the dyspnœa is very urgent do not perform tracheotomy unless it is due to bilateral laryngeal paralysis. If

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<sup>1</sup> The solids may consist of well-cooked meat or fish, and biscuit; and for the fluid 10 oz. of milk are permitted per day. From 12 oz. to 18 oz. solid may be permitted, but the fluid must not exceed 16 oz. It must be combined with absolute rest, and drugs are better avoided.



there be an external swelling some elastic support is needed. Calomel for high arterial tension ; aconite for palpitation. For venous distension or severe dyspnœa venesection may be performed. Surgical measures have been adopted from time to time in the treatment of superficial aneurysms, but they are not free from danger. Of such we may mention acupuncture, galvanopuncture, and the injection of coagulating fluids such as perchloride of iron (a dangerous procedure).

Lancereaux has recently introduced a novel method for the treatment of aneurysm by the hypodermic or intra-muscular injections of a solution of gelatin, and Dr. Robert Maguire informs me he has had considerable success with this plan ;<sup>1</sup> but Dr. Golubinin does not think this plan is successful.<sup>2</sup>

#### OTHER MEDIASTINAL TUMOURS.

§ 57. The **symptoms of mediastinal tumour** belong to three categories—namely, (a) the signs of displacement of organs ; (b) the physical signs of tumour ; (c) the symptoms of pressure. There are also (d) certain symptoms special to the different kinds of tumour.

a. The **displacement** of organs is sometimes the first intimation we receive. The liver is rarely displaced, but the lungs and heart are often pushed to one side ; and the apex-beat may be found in the axilla.

b. The **physical signs of tumour** appear sooner or later on the anterior or posterior aspects of the chest, and consist of (1) dulness on percussion, corresponding to the position of the tumour ; (2) auscultatory signs, which differ somewhat with the position and nature of the tumour. If it be solid, the breath sounds will be tubular, and there will be an increased conduction of the heart sounds. If it contain fluid (such as aneurysm, or more rarely, hydatid) there will be a diminished respiratory murmur, and in the case of aneurysm, a characteristic murmur (§ 56). (3) Ausculto-percussion will aid in defining the boundaries of the tumour. (4) Radiography is now coming into use for defining the nature and position of mediastinal growths.<sup>3</sup>

c. The symptoms of mediastinal tumour which are due to **pressure on the various structures around**, are as follows :—

(1) Dyspnœa always appears sooner or later, and may be of a type peculiar to mediastinal tumours, when there is pressure upon the trachea : it has a stridulous character, which resembles tubular breathing heard without the aid of the stethoscope. The breathlessness is often paroxysmal or asthmatic when there is pressure upon the heart and cardiac plexuses : or it may be of a Cheyne-Stokes nature. But the character of the dyspnœa depends upon whether it is the heart, the great vessels, the bronchi, or the nervous apparatus of the heart, lungs or larynx, which is pressed upon by the growth of the tumour.

<sup>1</sup> See Discussion Medical Society, *The Lancet*, Dec. 1, 1900.

<sup>2</sup> *Brit. Med. Journ.*, Oct. 13th, 1900, p. 60 (Épître).

<sup>3</sup> Dr. Hugh Walsham, *loc. cit.*

(2) Cough, sometimes of a laryngeal brassy character, is also present; and it is accompanied by expectoration if, as is usual, there is also bronchitis or congestion of the lungs. There may be laryngeal paralysis from pressure upon the recurrent branch of the vagus, and hoarseness or even aphonia may result from the same cause.

(3) Cardiac and circulatory symptoms, such as palpitation, or a difference in the pulses of the two sides in the neck or radial arteries.

(4) Dysphagia, from pressure on the gullet, is present chiefly with posterior mediastinal growths.

(5) Inequality of the pupils may appear, owing to pressure on the sympathetic. Usually the pupil on the affected side is contracted, from paralysis of the sympathetic, but it may be dilated when there is irritation of that structure.

(6) Pleuritic effusion occurs if there be pressure on the thoracic veins.

(7) The Inferior Vena Cava is rarely compressed, but lividity or œdema of the head, neck, and arms may occur from pressure on the Superior Vena Cava.

(8) In suspected tumour of the anterior mediastinum it is well to remember that when the head is thrown back the veins of the neck become distended, owing to the increased thoracic pressure producing venous obstruction.

(9) Pain down the arms and in the back occurs when there is pressure on the spinal nerve trunks.

d. *Causes.* There are certain symptoms which are special to the nature and situation of the tumour. There are five clinical groups of tumours in addition to aortic aneurysm.

I. MALIGNANT TUMOURS, which may be primary or secondary. If, in addition to the above physical signs, the expectoration present a constant prune juice character, and if on paracentesis a bloody fluid is drawn off, the presumption is strongly in favour of malignant tumour. Out of 520 cases of mediastinal tumour Hare found 134 were cancerous. Cancer of the mediastinum is the commonest mediastinal tumour, because it is usually secondary to cancer of the lung or œsophagus. In the latter case it is situated in the posterior mediastinum. Primary cancer, as of a bronchus, is rare, and tends to affect the anterior mediastinum. *Sarcoma*, especially lymphosarcoma, may start in the mediastinum as a primary growth. Sarcoma may also originate from the pleura and from the thymus remains. According to some, lymphosarcoma and Hodgkin's disease are identical, but this view is not now so widely accepted. Primary sarcoma is most frequent in the anterior mediastinum. If secondary in origin (as when the abdominal viscera are the seat of the primary tumour) it invades chiefly the posterior mediastinum. In primary mediastinal sarcoma enlargement of the glands in the neck and elsewhere may occur.

II. INNOCENT MEDIASTINAL TUMOURS, though more rare than the foregoing, are sometimes found in the mediastinum—e.g., fibroma, dermoid cyst, hydatid. Lipoma, gumma, and enchondroma, the latter growing from the sternum, are also occasionally met with.

III. ENLARGEMENT OF THE MEDIASTINAL GLANDS. With these there is often dulness posteriorly in the upper half of the interscapular space, but occasionally there is dulness over the sternum. Paroxysms of coughing, "croupy" or like whooping cough, may be present, together with stridulous breathing from pressure upon the trachea. The causes of enlarged bronchial glands are:—

(a) *Tubercle*, which is generally secondary to tubercle of the lungs. It is more common in children than in adults. The condition may be suspected when concurrent disease of the lungs is present and symptoms such as the above arise. If the glands suppurate, sweatings and intermittent temperature become more pronounced than when the lung only was diseased. An abscess may form and open into a bronchus (compare IV. below).

(b) *Lymphadenoma (Hodgkin's disease)* may start in the anterior mediastinal glands. Hare mentions 21 cases which he included under the names lymphoma (lymphosarcoma) and lymphadenoma. Lymphadenoma is much more rapid in its course, and cannot be diagnosed with certainty from lymphoma unless the ordinary symptoms of Hodgkin's disease be also present, viz., (i.) paroxysmal enlargement of the glands in other parts of the body; (ii.) paroxysmal attacks of pyrexia.

1 Hare [Mediastinal Tumours, Philadelphia, 1889] found out of 520 cases, 134 were cancer, 98 sarcoma, 21 lymphoma, 7 fibroma, 11 dermoid, 8 hydatid, 115 suppurative mediastinitis.

(c) *Bronchitis* and the *pneumonia* which complicates measles, influenza and whooping-cough, are often attended by enlargement of the bronchial glands; which, in children, may occasionally be recognised behind the sternum.

(d) *Whooping-cough*, without bronchitis or other disease of the lungs, may give rise to swelling of the bronchial glands, although the condition may be hard to make out. Some observers consider that it is the pressure of these glands which causes the paroxysms of whooping-cough.

IV. SUPPURATIVE MEDIASTINITIS (abscess of mediastinum) is a rare condition which may affect the ant. or post. mediastinum, or both, but more often the anterior. (i.) The most prominent symptom is pain, in the site of the inflammation, or passing down the nerves pressed upon. (ii.) Dulness, with edema and redness, may be present over the upper part of the sternum if the disease be in the anterior region, or over the dorsal spines if in the posterior mediastinum. Pulsation communicated from the aorta may be present and lead to a diagnosis of aneurysm, but the pulsation is not expansile, and fluctuation may be felt. (iii.) Pyrexia is present, usually of a hectic type (Chap. XVIII.), with the rigors, sweats, and weakness which attend all deep-seated inflammations. The causes of the acute form of mediastinitis are trauma, erysipelas, and the eruptive fevers. The chronic form is usually due to tubercular disease. It may rupture in various directions.

V. ENLARGEMENT OF THE THYMUS. A certain degree of enlargement is normal to childhood, and this may give rise to a dulness over the manubrium. In the rare cases, enlargement is met with in after life; it is generally due to carcinoma, and it may be a primary deposit.

*Prognosis.* In all cases of intrathoracic tumour which are large enough to produce symptoms the prognosis is unfavourable. Moreover, all of these conditions entail much suffering to the patient. Malignant tumours are fatal in 6 to 12 months, depending upon the site and progress of the growth. Innocent tumours may last for a long time. Syphilitic, tuberculous, and simple inflammatory glandular enlargements may recover under treatment, but even in these, no confident prognosis of recovery can be given in any case. Suppurative Mediastinitis may open externally, and run a course of a few days or weeks only; other cases are chronic and last for years, or lead to pulmonary gangrene and other serious complications when the pus burrows into adjoining organs.

*Treatment* in intrathoracic tumour is almost wholly palliative. For aneurysm see § 56. Abscesses, hydatids, or growths connected with the sternum may be dealt with by the surgeon in some cases.

## CHAPTER V.

### THE PULSE AND ARTERIES.

§ 58. **The meaning of "The Pulse."**—By the term "pulse" understood the expansile sensation communicated to the finger by the alteration in the shape of the artery, due to the momentary increase of blood pressure which takes place during the systole of the heart, and which is transmitted to the periphery *in the form of a wave*. It has been shown that there is no dilatation of the artery; the increased output of blood for the moment raises the blood pressure and alters the shape of the channel from an oval to a circle. The examination of the pulse is of extreme importance, not because it has a set of diseases of its own, but because it affords us so many valuable practical hints about the diseases of other organs, and about the general condition of the patient. "Many of the indications obtained from the pulse do not depend upon a comprehension of the circulatory conditions which the varieties of the pulse denote, or, indeed, upon a knowledge of the circulation at all. Observant physicians before the time of Harvey could gauge thoroughly the state of the patient in fever from the pulse, and it is not for the purpose of estimating the movement of the blood that we ourselves, in a case of fever, count the beats and note their force and volume. We calculate from the data thus obtained the strength of the sufferer, and the effect upon him of the disease. On the other hand, it is only through a knowledge of the conditions which govern the circulation that such facts as the connection between kidney disease and cerebral hæmorrhage can be understood, and that the prognostic significance of the hard pulse, which betrays this connection, can be appreciated."<sup>1</sup>

For the production of the pulse three factors are requisite :  
(i.) The contractions of the ventricle, which determine the

<sup>1</sup> Sir William Broadbent, "The Pulse," First Edition, p. 75. London, 1890.

frequency and rhythm of the pulse, and to a large extent its force; (ii.) the elasticity of the large vessels; (iii.) the peripheral resistance found in the arterioles and capillaries. These three factors must always be considered in studying the pulse.

§ 59. **Clinical investigation.** It is preferable not to examine the pulse until the preliminary excitement occasioned by the doctor's visit has subsided; and in all accurate records the pulse should be noted under similar conditions as regards the posture of the patient, time of day, relation to meals, etc.

The radial pulse is the one usually selected for examination, being easily accessible and lying against a bone. But the pulse can be observed in other situations—*e.g.*, the temporal, dorsalis pedis, or popliteal arteries. Three fingers should be placed along the course of the artery, the index finger next the heart, and allowance should be made for much adipose tissue. The different means of eliciting the several features will be dealt with below. When feeling the pulse its special features may often be brought out more fully by holding up both wrists with the fingers on the pulses. Only experience and comparison between all types of pulse can give to the physician the necessary aptitude for observation and correct inference. It is impossible here to enter upon all the complex data of the circulation, but the leading practical points which are of use in clinical work will be indicated.

A complete observation of the pulse should comprise six features, the first four being the most important.

1. *Rate and rhythm (i.e., regularity).* The rate of the pulse per minute is easily calculated by the watch, and in making this observation it should be remembered that a physiological acceleration occurs after any exertion, excitement, or after a meal, or even by the visit of the doctor. The pulse is faster in the evening than in the morning, and it is faster by about eight beats per minute in an upright than in a recumbent posture.<sup>1</sup> If the pulse be irregular, the type of the irregularity (*vide infra*) must be noted.

<sup>1</sup> The pulse is faster in the female than in the male, and it varies considerably at different ages, thus:

In the fetus and new-born infant its average rate is 140 per minute.

	Under 1 year	"	"	"	120	"
	" 3 years	"	"	"	100	"
From	7 - 14	"	"	"	90	"
"	14 - 21	"	"	"	80	"
"	21 - 65	"	"	"	70	"
	In old age	"	"	"	80	"



2. The *force* or strength of the pulse depends largely upon the force of the heart beats, and is best measured by its *compressibility*—the finger next the heart presses the vessel until the wave is no longer appreciable to the other fingers. By the amount of pressure required to obliterate the wave, the force with which the blood is propelled from the heart can be estimated. A “full bounding pulse” is one which has strong pulsations, but it is not necessarily one of high tension. Indeed, a full bounding pulse may occur in fevers where the tension is generally low. The *STRENGTH* of the pulse is measured by the force or *strength of the pulse wave*; the *TENSION* of an artery (*i.e.*, the b. p.) is measured by its condition *between the waves*.

3. The *character of each beat* is observed by noting (i.) whether the pulse-wave *rises* suddenly or gradually; (ii.) the *duration* of the beat, whether long or short; and (iii.) whether the *decline* is abrupt or gradual. It is important to note the presence or absence of *dicrotism*, which is a marked feature in low tension (see § 65).

4. The state of the *arterial tension* or blood pressure (b. p.) is perhaps the most important pathological feature of the pulse, and it is estimated by the degree of fulness of the artery *between the pulsations*. Normally the vessel is hardly felt between the beats, if the wall is healthy. The vessel should be rolled transversely under the fingers, and, if the arterial tension is high, it stands out like a cord *between the beats*.

5. The *size of the artery* and the *state of its walls* will require fuller consideration later on, but it is important to note these features, because an artery of small size may give the impression of a weak pulse. The thickness of the wall must also be noted, because a thick-walled artery may give the impression of high arterial tension.

6. The pulse of *both radial arteries* should be compared so that any abnormalities may be detected. It should be part of the routine to examine both pulses, as by this procedure we may detect the existence of unsuspected disease, such as aneurysm or other intrathoracic tumours. Abnormalities such as a more or less superficial position of the radial on one side or the other exist more frequently than is supposed.

It will be advisable to consider The Pulse under six headings, I. Infrequency; II. Rapidity; III. Irregularity; IV. Intermittency; V. High Arterial Tension (or Blood Pressure); VI. Low Arterial Tension. They are relatively of very different importance. Alterations of tension are of the greatest moment; and irregularity comes next. The **causes** of these will be considered; their treatment, with the exception of High and Low tension, belongs to the causal conditions.

The SPHYGMOGRAPH is an instrument employed to obtain a record on paper of the characters of the pulse. The first one used was that of Marey. In this instrument a pad placed over the pulse is connected with the short arm of a lever; the long arm, which magnifies the pulse wave, is sharpened to a point and makes a tracing on smoked paper.

The handiest instrument, however, is that of Dudgeon. This is a little instrument which is strapped on to the wrist. Some years ago Weiss

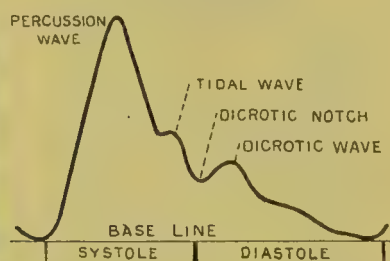
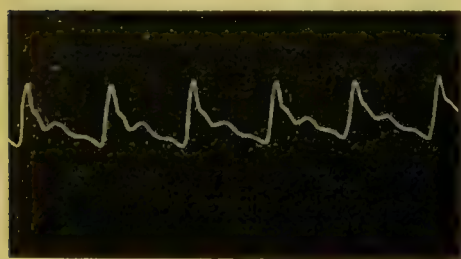


Fig. 23.—NORMAL PULSE TRACING, taken with the author's modification of Dudgeon's Sphygmograph. Rate, 68, pressure about 2 oz. Fig. 24.—NORMAL PULSE TRACING (Fig. 23) magnified, with the names of the principal parts. The dicrotic (or aortic) notch indicates the closure of the sigmoid valves, and therefore the termination of the ventricular systole and the commencement of the ventricular diastole. The diastolic line is that part of the tracing from the dicrotic notch to the next percussion wave.

made for me a modification of the latter which can be used without a strap—an appendage which I regard as unnecessary, since the instrument can more readily, with a little practice, be steadied and adjusted by the hand of the operator. The manipulation of any of these instruments is easily acquired by experience. The chief precautions are: (i.) to place the pad *exactly* over the artery, and it is of great assistance if the course of the vessel has been previously marked by an aniline pencil; (ii.) the *amount of pressure used*, and the adjustment of the instrument, should be such as to obtain the most graphic record.

The SPHYGMOGRAM or sphygmographic tracing is very useful as a graphic record of the pulse, and to show the progress of the case from day to day; but it does not tell us as much as the educated finger, and its readings can never be quite accurate, because the exact amount of pressure exercised by the pad upon the artery cannot be known.

Figs. 23 and 24 represent a normal pulse tracing—the principal named parts of which it consists being indicated in the latter. 1. The *percussion wave* is abrupt and vertical in proportion to the force of the ventricular



Fig. 25.—Oliver's Hemadynamometer.

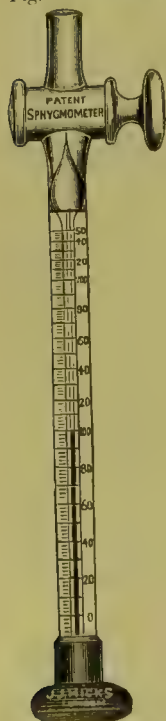


Fig. 26.—Hill and Barnard's Sphygmometer.

contraction, and inversely proportional to the peripheral resistance. 2. The *tidal wave* is prominent in proportion to the amount of peripheral resistance and the force of the ventricular contraction; *i.e.*, when the arterial tension is *high*. When the peripheral resistance is very low there may be no tidal wave. 3. The *dicrotic wave* is pronounced and the *aortic notch* more marked in proportion as the peripheral resistance and the heart force (*i.e.*, the arterial tension) are low.

The HEMADYNAMOMETER (Fig. 25) is an instrument devised by Dr. George Oliver to measure the *blood-pressure*, and he has also invented an ARTERIOMETER (Fig. 32a) for measuring the *internal diameter* of the radial artery. A description of their method of use is sent out with them. There are three precautions which it is well to bear in mind in the use of these instruments: (i.) the pad must be placed *exactly* over the radial artery, and to this end the position of the latter should be previously marked very pre-

cisely with an aniline pencil; (ii.) several readings must be made and only the lowest of these should be taken (whether for the size of the artery or for the blood pressure), because all errors are on the side of excess, and are generally due to the non-adaptation of the pad to the artery; (iii.) considerable practice is indispensable, and one cannot hope to become proficient in the use of these instruments without using them frequently for several weeks.

Fig. 26.—HILL and BARNARD'S SPHYGMOMETER is an instrument designed to measure the blood-pressure. It is used as follows: First open the tap at the top and adjust the top of the spirit index to 0 on the scale, then shut the tap. Having marked with an aniline pencil the position of the artery, adjust the indiarubber pad of the instrument exactly over the vessel in the position, and with that degree of pressure, which obtains the maximum excursions corresponding to the pulse. By steadily increasing and regulating the pressure of the disc upon the wrist it will be found that with light pressure and similarly with heavy pressure the movements of the index indicating the pulse become less. But there is a position midway between the two at which the movements of the fluid are greatest. This middle position, in which the rise and fall of the fluid with the pulse is greatest, indicates the blood pressure.

When the artery is superficial, like the radial, and the walls are normal, the readings have seemed to me quite reliable. But when an artery is deeply seated, or when its walls are rigid, a fallacy is introduced which renders the results erroneous and even worthless. The average b. p. in the radial artery is 100, which represents a pressure of 100 mm. of mercury. Hill's investigations with the above instrument have shown (Proc. Physiol. Soc. Jan. 15, 1898), that exercise raises the b. p. considerably—for instance, after running 400 yards as fast as possible, the b. p. would rise from 120 to 130. Posture also influences the b. p., but only when the person is not fatigued. Thus in the morning, in a horizontal position, the average pressure was 105, and 120 in a standing position. But in the evening, both horizontal and vertical postures gave a reading of 105 to 110. The b. p. at any moment depends upon many circumstances; and amongst other things an individual who has been trained to a certain exercise will not exhibit the alterations of pressure which would be found in an untrained person.

HAIG'S CAPILLARY DYNAMOMETER will be described in Chap. XVII.

§ 60. **Infrequent Pulse**, Slow pulse, or Bradycardia (*i.e.*, slow heart), is met with under several conditions. In *health* a slow pulse is normal to certain individuals without any very obvious explanation, and without any other symptoms. Napoleon is said to have rarely had a pulse-rate over forty. I have known a gentleman for twenty years, whose age is now eighty-two, who has never, to my knowledge, had a pulse-rate over fifty when in health; its average is forty. He has always enjoyed very good health. In some families it is met with as a hereditary condition. It is always well to verify an apparently slow pulse by listening to the heart at the same time, for in some cases the infrequency of the pulse may be due to some of the heart beats not reaching the wrist. *Pathologically* a slow pulse is of importance chiefly in heart and brain disorders.

1. In *heart disorders* a slow pulse without irregularity is uncommon. It may however be met with in connection with the senile heart, where it always indicates some degree of dilatation. Sclerosis of the coronary arteries, atheroma of the aorta, and fatty or other degeneration of the cardiac wall, may also be attended by bradycardia.<sup>1</sup>

2. If a slow pulse is associated with marked high tension *arterial sclerosis* should be suspected even when no sign of arterial disease can be discovered.

3. Various *gastric derangements* are frequently associated with a slow pulse, probably in a reflex manner. This is, perhaps, the commonest cause of slow pulse in children. In adults it may be associated with gastric ulcer and carcinoma. In chronic dyspepsia a slowness of the pulse is usually associated with some amount of irregularity and intermission.

4. Many *nervous disorders* may be associated with slow pulse. Thus:—

(i.) If the temperature is raised, the arteries contracted, and the pulse slower than normal, and if with this there be some irregularity in rhythm, and perhaps a reduplicated second sound, in a child, we probably have to do with an early stage of meningitis.

(ii.) Cerebral tumour is another nervous condition associated not infrequently with slow pulse. Here, however, it probably only occurs in the late stages and as a pressure symptom. Halberton mentions a case in which a violent blow on the head was followed by a permanently slow pulse, with syncopal attacks, succeeded by epilepsy; the post-mortem revealed narrowing of the foramen magnum.

(iii.) Various psychical disorders, such as melancholia, general paralysis, and epilepsy, may also be attended by slow pulse.

(iv.) An abnormally slow pulse has been observed in association with the following

<sup>1</sup> When bradycardia is associated with epileptiform attacks, some, *e.g.*, Dr. J. W. Ogde (*Lancet*, January 30th, 1897, "Unusual and persistent slowness of the pulse"), are of opinion that it indicates disease, involving the base of the heart, and irritability of the cardiac ganglia situated there. Convulsive attacks may occur with any cardiac enfeeblement, whether due to gross disease at base of heart or degeneration of its muscular wall, and are doubtless accounted for by cerebral anaemia.



spinal lesions especially affecting the medulla and cervical regions:—fracture of the fifth and sixth cervical vertebræ was followed by a permanently slow pulse beating 48 to the minute (Hutchinson); fracture between the cervical and dorsal regions was followed by a pulse of 20 to 36 per minute (Gurtl); a violent blow over the sixth cervical vertebræ in a child was followed by a pulse of 48—56 per minute (Rosenthal).

5. *Drugs* such as digitalis and strophanthus slow the rate of the heart, and, if irregular, steady its rhythm. Belladonna and tobacco at first slow the heart.

6. Slow pulse is also met with in *toxic* conditions, such as diabetes, jaundice, uræmia, and poisoning by carbon monoxide.

7. In states of prolonged *exhaustion* and anæmia, and in *convalescence* from acute illness, bradycardia occurs.

§ 61. **Quick Pulse.** The rapidity of the heart beat varies considerably within the range of *health*, and in many persons the heart may at times beat 150 a minute for a short time without inconvenience. In infancy the normal rate is 130, and this may continue in after life.<sup>1</sup> The pulse is normally more rapid during the menstrual period and menopause, in the evenings and after meals. After a severe illness the pulse more easily becomes rapid.

The *pathological* causes of quick pulse are numerous. Apart from cardiac affections, or Graves' disease, a quick pulse is relatively unimportant in the young. In general terms it is only a serious symptom when met with in the latter half of life. It is very desirable in such cases to obtain a sphygmographic tracing, for the danger of a quick pulse may be fairly measured by the amount of diastolic present.

1. In diseases of the *valvular structures* of the heart the pulse is quickened, more especially in mitral and aortic regurgitation; and also in the later stages of all forms of valvular lesion when cardiac failure commences. In extreme dilatation there is sometimes a condition known as *delirium cordis*, where irregularity and rapidity of action are combined. Fœtal rhythm is also met with in dilatation. (See Heart, § 47.) An *insidious chronic endocarditis*, before the occurrence of a murmur, may be evidenced by heart hurry as the only symptom for months. This is especially the case in mitral stenosis; Balfour<sup>2</sup> mentions a case in which the presystolic murmur appeared two years after the heart hurry commenced.

2. In *cardiac dilatation* (apart from valvular disease) the pulse

<sup>1</sup> See footnote, p. 125.  
<sup>2</sup> "The Sentle Heart," 1894.



is quickened, especially if accompanied by arterial atheroma or sclerosis. In the latter half of life heart hurry is nearly always associated with dilatation of the heart, the result of a degenerative change. Thus, one of the earliest signs of the failure to compensate for the obstruction caused by arterial disease, is a persistent tachycardia. Quick pulse indeed is regarded by some (*e.g.*, Balfour, *loc. cit.*) as the leading feature of both "irritable" and "senile heart."

3. Various *toxic conditions* increase the heart rate. The heart hurry which attends chronic alcoholism is very serious, as indicating fibroid or fatty degeneration of the heart wall or neuritis of the vagus. Tobacco first slows the heart, but in large doses paralyses the vagus; hence excessive smoking may induce paroxysmal tachycardia. Digitalis in large doses also paralyses the vagus (Balfour). Belladonna increases the rate, fullness and force of the beat, and increases the blood pressure (Balfour), but in toxic doses it paralyses the vagus and produces tachycardia. Tea and coffee may produce temporary heart hurry.

4. Many *affections of the nerves*, functional and organic, are attended by heart hurry, usually transient, but sometimes persistent. Emotional rapidity of the pulse is familiar to every one. Paroxysmal tachycardia is described elsewhere (§ 43). Tumours pressing on the vagus may be evidenced by heart hurry, even if the tumour be small; a rapid pulse may indeed be the only symptom.

5. In *Graves' disease* heart hurry is often the earliest symptom, and in this disease there may be throbbing in the whole arterial system, the heart sounds being clear and distinct.

§ 62. **Irregular Pulse (Arrhythmia).** In *health* irregularity is very rare, excepting in old age and under the influence of transient emotion (mental or bodily shock). In nervous subjects it may occur, as the result of tea, coffee, or tobacco in excess. In *diseased states* the pulse may be irregular in rhythm, in force, and in volume. Irregularity of volume or force is of more importance than irregularity of rhythm. The prognosis of irregularity is less favourable than that of intermission. In acute diseases irregularity of the pulse is a bad omen, because it indicates cardiac exhaustion.

1. Irregularity is chiefly found with *cardiac valvular disease*, and constitutes one of the leading indications of *failing compensation*. In mitral stenosis it sometimes occurs even while compensation is perfect; but irregularity of the pulse is especially marked in the failing compensation of mitral regurgitation. As a point of distinction we may remember that the tension in mitral stenosis is relatively high, in regurgitation it is low.

2. Irregular pulse, in the *absence of all cardiac lesions*, is usually the result of some reflex disturbance of gastric origin. This may occur after a heavy meal, or in association with a distended colon, either of which may cause irritation of the vagus terminations, or produce a direct pressure upon the heart. Any mechanical impediment to the heart may produce irregularity, and this may be the explanation of the frequency with which irregularity attends emphysema. In this case the expiratory may differ from the inspiratory pulse rate; the former being the more rapid. Sometimes chronic gout, and sometimes atheroma, are attended by an irregularity of the pulse.

3. *Fatty or fibroid heart*, without valvular disease, is often evidenced by irregularity. If the pulse becomes regular after a little exercise there is not much amiss with the heart-wall, but if the irregularity is increased it indicates that there is serious damage.

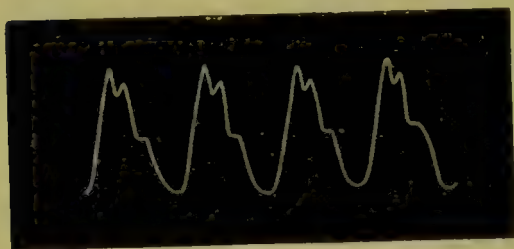


Fig. 27.—PULSUS BISFERIENS. I. G., æt. 70, slight aortic stenosis. This tracing is not a good illustration; the 2nd wave ought to be higher than the first.

In *pulsus paradoxus* there is complete, or almost complete, disappearance of the pulse during inspiration. It has been shown by Dr. Maguire<sup>1</sup> to be due to either (1) an increase of the "negative" intrathoracic pressure which normally takes place at the end of inspiration, or (2) an extreme weakness in the left ventricle, or to both. Maguire has shown that it can be produced in even healthy persons at the end of inspiration by so contriving that the negative intrathoracic pressure can be *suddenly* increased. It is met with (due to 1) in intrathoracic tumours.

The *pulsus bisferiens* (Fig. 27) is a rare and peculiar type of pulse occurring in some cases of aortic disease (stenosis or regurgitation and especially double). It is apt to be confused with the dicrotic pulse, though it indicates neither increase nor diminution of tension. A double beat is felt because the ventricle makes two distinct efforts during the systole. It is distinguished from dicrotism by becoming more evident by pressure, whereas the dicrotic pulse is obliterated by pressure.

The *anacrotic* pulse is a somewhat rare condition, resulting from high arterial tension. In it the tidal wave is higher than the percussion wave. It is found in some cases of aortic stenosis and aneurysm, where the ventricle has to overcome abnormal resistance, and the blood cannot rush out in full volume during the systole.

<sup>1</sup> "On the Dynamics of the Thorax in Disease." Dr. Robt. Maguire, *Clinical Journal*, August, 1898.

The following special kinds of irregularity are found chiefly in mitral disease, and especially in mitral disease under treatment by digitalis.

(a) In the *pulsus myurus*, a rare form, there is one strong beat followed by a succession of beats of diminishing volume.

(b) In the *pulsus alternans* there are two beats of the heart to one of the pulse, and it may be that the second is hardly perceptible at the wrist.

(c) In the *pulsus bigeminus* the beats come in couples, a strong beat being followed quickly by a somewhat weaker one, after which there is a pause. On auscultation the heart's action may be found to correspond with this irregularity, but sometimes an abortive contraction occupies the position of the missed beat. Examination of the heart in *pulsus bigeminus* gives the impression of a contraction only of the right side of the heart during the abortive beat (Broadbent, *loc. cit.*). In the *pulsus trigeminus* there are 3 beats and a pause.

§ 63. In the *Intermittent pulse*, which is of less importance than irregularity, a beat is missed at intervals, after several (say 5 to 20 or 30) regular beats. On auscultation the heart is found sometimes to pause entirely as the pulse does; but more frequently it makes a rapid and imperfect contraction corresponding with the missed beat at the wrist. No symptoms may be experienced by the patient, but sometimes there is cardiac discomfort, or palpitation. If intermission be due to a gastric or nervous cause, the patient is as a rule conscious of it; if, on the other hand, it be associated with a cardiac condition, he is more often unconscious of it. Intermission is of minor importance if it can be made to disappear with exercise.

In *health* intermission may be found either as a constant feature, or after meals, and in these circumstances the pulse is restored to normal during exercise, fatigue, excitement, and pyrexia. In some people tea, coffee, or tobacco will produce intermission.

1. Intermission of the pulse may attend *cardiac valvular disease*, but it is rare unless associated with irregularity (*vide supra*).  
2. It also occurs with fatty heart and in other *lesions of the wall* (see "Irregularity"); but (3) intermission is more frequently due to gastric derangement, and occurs most often after a heavy meal. It is fairly common in chronic gout.

§ 64. *High arterial tension*<sup>1</sup>—i.e., high blood-pressure—is undoubtedly, when habitual, the most serious of the pathological conditions of the pulse, because of the continuous strain it produces upon the cardio-vascular system. Normally the artery collapses completely beneath the finger between the beats; but if the arterial tension or blood-pressure be too high *the artery*

<sup>1</sup> It has become a common practice to speak of "pulse tension," "high-tension pulse," etc. It is not correct to say that a wave has pressure or tension, but the terms have become sanctioned by custom.

remains full between the beats, and sometimes stands out like a cord, so that it can be rolled under the finger. In applying this test we must beware of the fallacy of a thickened artery, also the "recurrent pulse"—i.e., a wave flowing back from ulnar to radial artery. This may give the sensation of a radial pulse hard to compress, but will disappear on pressing the third finger firmly over the radial artery, at a point distal to the first finger. The pulse-beat or wave of high tension *may* appear small, because when the artery is constantly and fully distended, it cannot be much more distended by the pulse wave. This is revealed in a sphygmogram (Fig. 28),

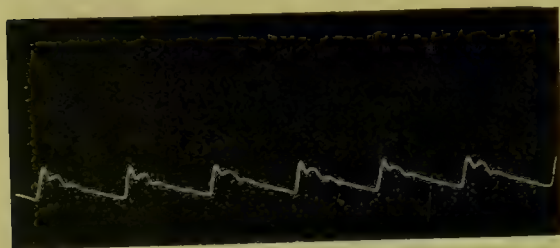


Fig. 28. - HIGH TENSION in a case of Chronic Bright's Disease with Albuminuric Retinitis, under the care of Dr. J. S. Bristowe. High tension is indicated graphically by—1, prominence of the tidal wave (unless the arteries are very atheromatous); 2, smallness of the diastolic wave, and its occurrence high up on the diastolic wave, and its gradual sloping of the diastolic line towards the next upstroke.

by the small extent of the excursion made by the lever. In high tension the upstroke is short and sloping, and the percussion wave tends, it will be seen, to be flat-topped. The rate is not generally increased.

VIRTUAL TENSION is a term applied by Broadbent to a condition of pulse due to *rigidity of the aorta*, which simulates high tension. In consequence of this rigidity the systolic shock is transmitted unmodified to the periphery, and the distension of the radial artery is *at first* maintained as in true high tension, though only for a fraction of a second; then it suddenly collapses. It will be best understood by consulting the sphygmogram (Fig. 20, B, p. 108; and Fig. 33, p. 145), where the percussion wave is seen to be flat, or, rather, square-topped. The first-half only, or one-third, of each beat is in a state of high or sustained tension: the remainder is low-tension. Virtual tension is the leading feature of the *senile pulse*, since in the aged a rigid aorta is more or less normal.

The *symptoms* which accompany high arterial tension, and which lead us to suspect that condition, are very important, though somewhat variable. They consist of (i.) headache, which may be frontal, occipital, or vertical, accompanied by vertigo from time to time, and a constant feeling of fulness about the head. Sometimes the headache assumes the character of migraine, sometimes of neuralgia. (ii.) There may be some lassitude, disinclination for exercise, and depression of spirits. (iii.) Breathlessness



on exertion is common; very often it is paroxysmal, and the patient thinks he has asthma. (iv.) Wakefulness, or sleeping by dozes, is not uncommon.

High tension is rare in children, but these symptoms occurring in adults, especially in those past middle life, are suspicious, and are confirmed if, upon examination, we find the following physical signs: (i.) The pulse revealing the above characters. (ii.) On auscultation an accentuated aortic second sound (sometimes accompanied by a reduplicated first sound at the apex). (iii.) Later on, if the condition persist, or constantly return, cardio-vascular hypertrophy supervenes.

The CAUSES of high arterial tension are numerous and of considerable importance. Among the *predisposing causes* heredity undoubtedly plays a most important part. "No condition, indeed, runs more strongly in families than high arterial tension, and it is the explanation of a family liability to apoplexy and paralysis, or to death from heart disease" (Broadbent, *loc. cit.*). No age is exempt from high arterial tension, but it is found far more frequently at and past middle life. As regards sex, males are more subject to the disease, since they are more exposed to the dietetic and other influences which raise arterial tension.

*Exciting* causes bring into operation one or more of three pathological factors—increased peripheral resistance, increased cardiac force, or increase in the volume of the blood. The exciting causes are as follows, arranged fairly in order of importance:—

1. Anything in the mode of life which leads to *deficient oxygenation* and *elimination of nitrogenous waste*, such as: (i.) excess of nitrogenous food—meat, and even poultry and game, soups, beef-tea, and other meat extracts and preparations containing extractives. The condition is believed to be more common among meat-eating races. (ii.) Alcoholic drinks of any kind, even in moderation. (iii.) Sedentary habits, or any mode of life leading to deficient exercise in the open air. (iv.) Constipation is another very important cause. All of these four causes lead to increased resistance in the capillaries in the opinion of some observers,<sup>1</sup> by producing a toxic condition of the blood which acts either directly on the capillary walls, so provoking their contraction, or by causing cohesion of the blood to the capillary wall. Constipation may act also in another way—viz., through its effect upon the abdominal circulation, for it drives the blood out of the large abdominal veins into the general circulation, and so raises the general blood pressure. This is the current explanation, but it seems to me that the toxæmia which results is far more likely to act by irritating and so constricting the peripheral arterioles.

2. Several *toxic conditions* of the blood act probably in the same way. (i.) Renal disease is a most familiar antecedent and accompaniment of high arterial tension. The renal diseases here referred to are those which result in inflammation or degeneration of the parenchyma—acute and chronic desquamative nephritis, and granular kidney—i.e., those which

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<sup>1</sup> e.g., Sir William Broadbent, *loc. cit.*



lead to the imperfect elimination of nitrogenous waste. (ii.) Gout, where there is probably excessive retention of nitrogenous waste. High arterial tension so frequently accompanies this condition that it is known sometimes as the "gouty pulse." (iii.) The diabetes which occurs in persons past middle life is attended by high tension, but not that more fatal form of diabetes which occurs in younger subjects. (iv.) Plumbism, which is intimately connected with gout and renal disease. (v.) Emphysema and sometimes other lung conditions (probably by deficient oxygenation). (vi.) Anæmia, sometimes. (vii.) Pregnancy is invariably accompanied by increased tension, probably on account of the hyperinotic condition of the blood.

3. Any condition leading to a *persistent contraction of the arterioles* results in high arterial tension. It is highly probable that some of the above toxic conditions may act in this way. Migraine, which is attended by constantly recurring spasm of the peripheral vessels, thus comes to be attended by high arterial tension. One of the results of high tension is an increase in the muscular coat of the vessels, and this in turn tends to perpetuate constant high tension.

4. *Plethora*, by increasing the volume of the blood, necessarily results in increased arterial tension.

5. *Cardiac valvular disease* is more often attended by a diminution than an increase of blood pressure. Mitral stenosis is almost the only variety with high tension; though we may get all the features of high tension in aortic stenosis. It is, however, in *cardiac hypertrophy* that we have the most constant tendency to high tension. Whenever the heart beats more rapidly and more powerfully, as for instance during excitement or during exertion, more blood passes into the vessels in a given time, and there must necessarily be a rise of pressure. This kind of high tension is usually transient, but if frequently repeated, as in athletes, it may be a forerunner of a persistent high arterial tension.

6. In certain *neuro-vascular* diseases there is a tendency to dilatation, and in others to spasm, of the peripheral vessels. In the latter (*e.g.*, Raynaud's Disease) there is a constant tendency to high tension.

*Pathological effects of high tension and prognosis.* Temporary high arterial tension has no serious consequences, but when the increased tension extends over many months or perhaps years, it has very grave ulterior results. By the physiological law that increased function results in increased growth, there is hypertrophy of the muscular tissues of the whole of the vascular system, that is to say, hypertrophy of the heart and of the muscular tissues of the arteries (arterial hypermyotrophy, § 71). This may be said to constitute the first stage. If the increased tension continue, a degeneration occurs in the muscular tissue of the heart and of the arteries. The heart muscle gradually degenerates and its cavities dilate. The muscular tissues of the vessels undergo first a granular degeneration (the early stage of sclerosis), which is attended by hardness, loss of elasticity and of contractility. The second stage is

chiefly manifested by the failure of the heart to compensate for the increased peripheral resistance caused by the rigidity of the arteries. Moreover, on account of the changes in the vessels, rupture of the peripheral vessels is a common consequence; and as the vessels of the brain have least support they are the most frequent seat of rupture. Atheroma and loss of elasticity of the aorta are other consequences of persistent high tension. (See § 69.)

The subject of *prognosis* will now be more readily understood. People with increased tension may live for a long time, but there is a constant liability to the accidents above mentioned, and they certainly do not live so long as those without high tension. The general habits and temperament of the patient greatly influence the prognosis, both as regards the removability of the causes and the liability to accident. Where the heart is hypertrophied the prognosis is not serious so long as compensation does not give way, for the progressive changes can be arrested by lowering the tension and improving elimination. Even in chronic Bright's disease proper treatment may prolong life for a considerable time. In hereditary cases, some light may be thrown upon the prognosis by inquiry as to the ages and causes of death of the ancestors. The prognosis is good in general terms where the causes are removable, such as constipation, temporary lithæmia or plethora, and in cases where the tension is brought down by the administration of salines. On the other hand, it is grave in advanced renal disease, alcoholics, and those who will not or cannot adhere to treatment. As regards the untoward symptoms a ringing accentuated second sound (indicating dilatation of the aorta) or excessive tortuosity and beading of the vessels (indicating advanced vascular disease), are of unfavourable import. Dilatation of a valvular orifice secondary to vascular strain is more serious than primary valvular disease of the heart, because in the latter case the weakened heart has not the extra work of overcoming the vascular resistance.

*Treatment.* The two main indications are to keep the blood free from impurities and to relieve the strain on the circulation. General hygienic measures occupy a very prominent position. Moderate regular exercise and plenty of fresh air, with a minimum of nitrogenous food, and the avoidance of alcohol, are cardinal

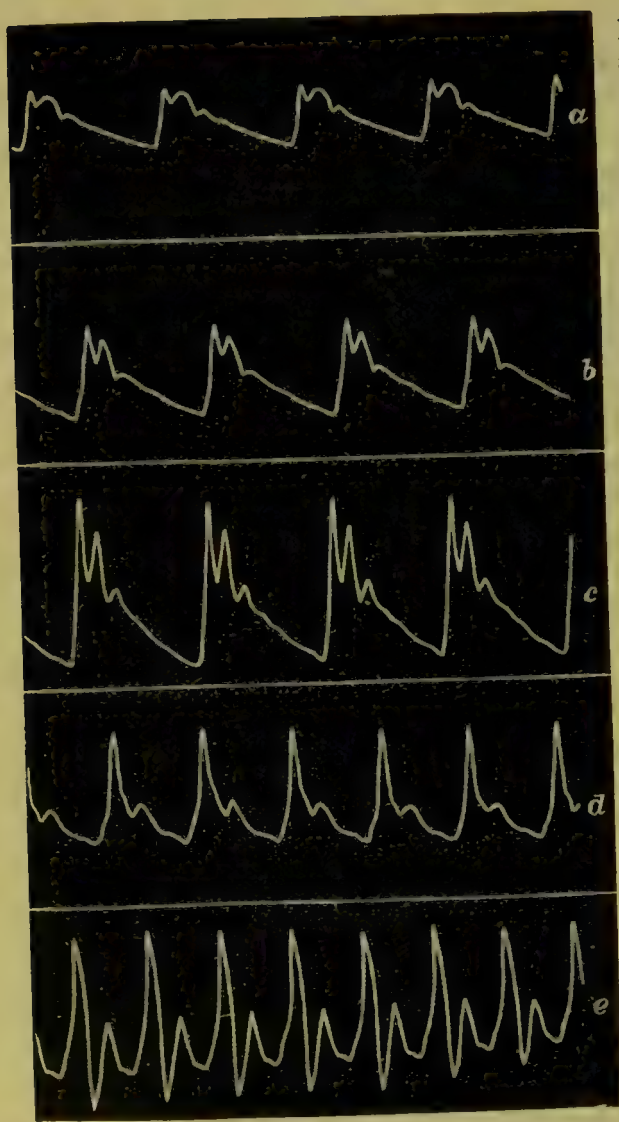


Fig. 29.—HIGH TENSION PULSE REDUCED to a state of hyper-dicrotism (converse of Fig. 32).—Series of tracings showing the efficiency of sodium nitrite in the reduction of high tension. Martin R., *et. 45*, Chronic Parenchymatous Nephritis, under the care of Sir Wm. Gairdner, in the Western Infirmary, Glasgow, August, 1895. *a*, Typical high tension. Tracing *b* shows the reduction of tension after 15 grs. sodium nitrite in 4 doses during the 24 hours. The remedy was continued, and tracings *c* and *d*, on successive days, show the gradual reduction of tension effected. They are normal excepting for the exaggerated aortic wave. In the last tracing (*e*) a condition of HYPER-DICROTISM is shown, the high tension having been entirely replaced by the opposite extreme. (Kindly supplied by Dr. W. S. Cook, who was the House physician at the time.)

principles. Meat should only be taken once a day (§ 183); copious libations of water aid elimination, and great benefit may be derived from the Turkish bath. Purgatives should be regularly administered, especially salines, such as Carlsbad salts, mag. sulph., mag. carb., phosphate of sodium, one or two drs. every morning. A mercurial purge once or twice a week may be given. Great value is attached to mercury; the tension may nearly always be reduced by the administration of one or two grs. of calomel, or pil. hydrarg. It may be combined with pulv. ipecac. ( $\frac{1}{2}$  gr.), rhubarb, or colocynth. Heat, either in the

form of radiant heat, hot-air baths, or hot water, dilates the cutaneous vessels and thus removes the peripheral resistance sometimes in an almost magic manner. This and liq. trinitrin will remove the headache of high tension. The nitrites and other vaso-dilators are very useful to reduce tension (see Figs. 20, 21, and 29). If the vessels are already the seat of sclerosis, the remedies advised (§ 70) should be employed; and some care is required in the administration of baths.

§ 65. **Low Arterial tension.**<sup>1</sup> In low arterial tension the artery cannot be felt between the beats; the pulse comes rapidly up to the finger and rapidly declines, and it is very easily obliterated. In very low tension a double wave is felt, the second one being very small; this is known as the *dicrotic pulse*. A dicrotic pulse indicates very low arterial tension. A sphygmographic tracing shows an increase of the normal depression



Fig. 30.—LOW TENSION PULSE-TRACING from a case of intestinal obstruction.

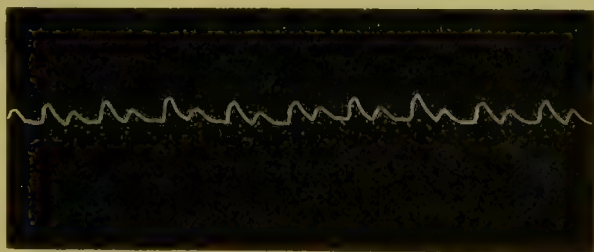


Fig. 31.—LOW TENSION PULSE-TRACING from a case of Enteric Fever, showing marked dicrotism. Patient æt. 37.

(aortic notch) before the dicrotic wave, and the dicrotic wave itself is more marked than in a healthy pulse (Figs. 30 and 31). The dicrotic wave may be as high as the percussion wave, and thus there may appear to be two beats for each cardiac pulsation. When the aortic notch falls below the level of the base line the pulse is said to be *hyperdicrotic* (Figs. 29 and 32). *Symptoms* of depression, lassitude, prostration, and sometimes dyspepsia and sleeplessness, are found in association with low arterial tension. Purgatives cannot be well borne; and the patient may feel better when the bowels are

<sup>1</sup> Low tension pulse is a term frequently used, but the same objection applies to it as to the term high tension pulse—viz., that a wave cannot be said to have tension or pressure.



constipated, because the retained fæces drive the blood out of the splanchnic area of veins, and so raise the general blood pressure. Capillary pulsation is sometimes met with in low arterial tension, particularly with aortic regurgitation. By drawing a line along the forehead, or lightly pressing down the tip of the nail, the alternate blush and pallor due to the capillary pulsation is well brought out. In extreme states a pulse may even be communicated to the veins on the dorsum of the hand.

*Causes.* In *health* a persistent state of low tension is sometimes, though rarely, a hereditary condition. It may be found also after meals, a warm bath or moist warmth.

1. With *cardiac valvular disease* in all phases of failing compensation there is low tension. The pulse of aortic regurgitation is so characteristic that the lesion can be diagnosed from it. It comes

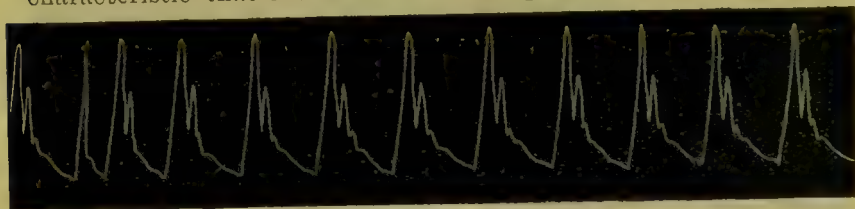


Fig. 31A.—WATER-HAMMER PULSE.—Tracing (taken by Dr. Reginald E. Hanson) from a man, æt. 34, with typical aortic regurgitation, accompanied by very great cardiac hypertrophy (bovine heart). Well-marked capillary pulsation and retinal pulsation were present. Typical collapsing or water-hammer pulse. He had had anginoid attacks, which were relieved by sodium nitrite. At the time tracing was taken pulse was 81, respiration 22, and blood pressure (taken by Hill and Barnard's instrument) 140.

suddenly up to the finger, and as suddenly collapses. This has received the name of “shotty,” “slapping,” “water-hammer” or “collapsing” pulse, the pulse of unfilled arteries, or Corrigan's pulse, after the name of its first describer. It is best brought out by feeling the radial while the patient's hand is held vertically up on a level with the head. In this condition the radial pulse is distinctly postponed, that is to say, the pulse at the wrist occurs later than the apex beat. Moreover, the artery in aortic regurgitation is large, in contradistinction to aortic stenosis, where the artery is small. The sphygmographic tracing of the “water-hammer” pulse shows a long percussion stroke, scarcely any tidal wave, with a sudden down stroke, broken only by a small diastolic wave (Fig. 31A). In cases of a double aortic murmur the pulse is of great diagnostic significance. If the pulse has a distinctive “water-hammer” character, the systolic murmur is due not to aortic stenosis, but

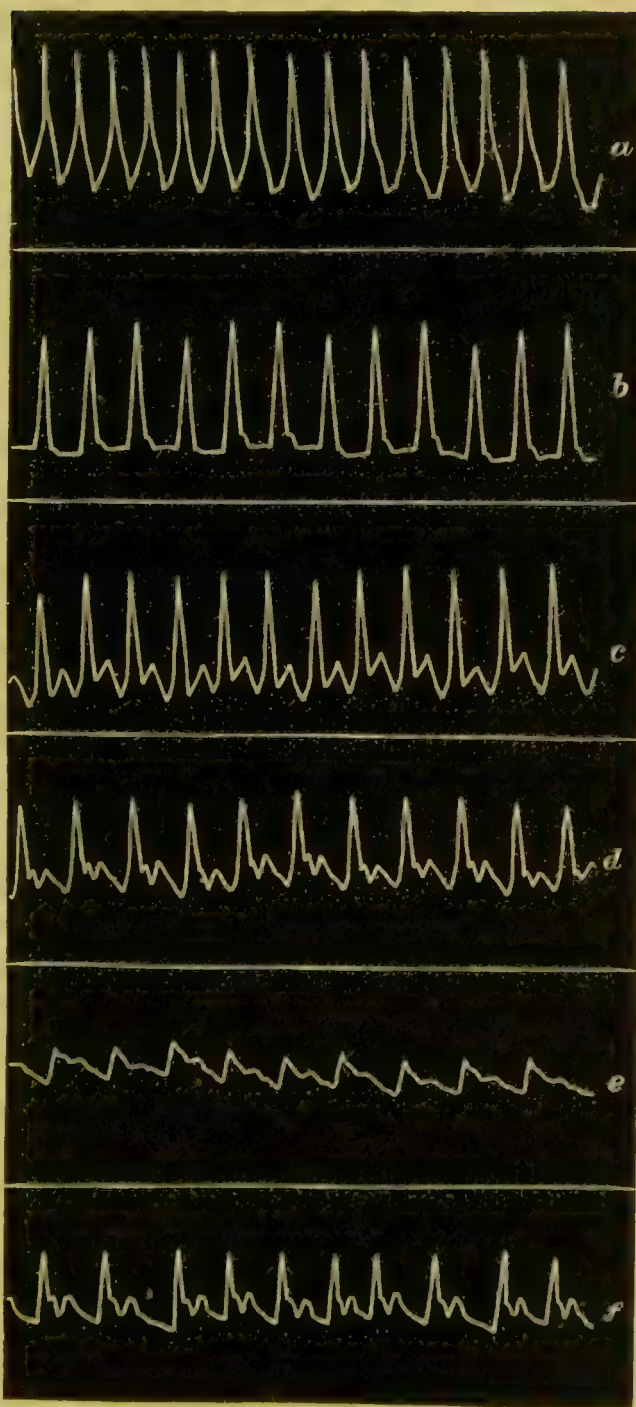


simply to roughening of the valves or atheroma of the aortic wall.

2. In *weak heart* — *e.g.*, fatty heart, and general obesity — the pulse is of low tension.

3. Without organic heart disease low tension is met with in neurasthenia and in all STATES OF

Fig. 32. — HYPERDICTOTISM of low tension gradually raised to normal (converse of Fig. 29). The author is indebted to Dr. W. S. Cook for the notes of the following case, which was under Professor Gairdner's care in the Western Infirmary, Glasgow. Reginald R., *æt.* 15, was admitted on June 5th, 1895. The patient was attacked with ACUTE DOUBLE PNEUMONIA 2 days before admission. The dullness had a scattered character, which aroused a suspicion of tuberculosis, but examination of the sputum showed an absence of tubercle bacilli, and a great abundance of Friedländer's pneumococcus. Great prostration and delirium were marked features in the case. The tracings show very well the hyperdictotic, *i.e.*, low tension pulse at the commencement of the illness, and the gradual assumption of a normal tension in convalescence. The treatment was as follows: On the 5th June, 6 gr. quinine every 3 hours; on the 6th, a mixture of *vin. ipecac.*, *sp. æth. nit.*, and *liq. ammon. acet.*, with 2 gr. quinine every 3 hours; on the 7th, 5j. whisky every hour; on the 9th, ice-cloths to the abdomen, and paraldehyde at night; on the 11th the ice-cloths were discontinued.



EXHAUSTION and debility, such as are caused by over-exertion, physical or mental, deficient and bad food or anxiety—all conditions in which vaso-motor paresis occurs. Low tension is also met with in asthenic varieties of fever, especially enteric fever and peritonitis (see also Fig. 32).

*Treatment* of course depends upon the cause. In general terms, the food should be nourishing, and easily digestible. Change of climate is often beneficial. Iron, arsenic, quinine, strychnine, and digitalis are all useful; and alcohol, which should, however, only be given with meals and in small amounts. It is, nevertheless, very useful then in the aged. Fig. 32 shows the raising of low arterial tension under suitable treatment.

**§ 66. The pulse in relation to prognosis and treatment of disease.** In acute diseases of a febrile type a full bounding pulse is natural, and its absence warns us that we are in the presence of an asthenic and more dangerous type of case. It is in fevers also that the pulse is our chief guide as to whether alcohol should be administered or not. In enteric, for instance, if the pulse is weak and the vital powers flagging, stimulants are called for, but not otherwise; this was Murchison's rule.

In chronic affections the pulse is not so valuable an indication of the patient's general condition. Perhaps the most important fact to bear in mind is that in the aged, and, indeed, all persons past middle life, the pulse should be carefully watched from time to time, because a constant high tension is the main cause of arterial degeneration, and many of the most lethal diseases to which persons in the latter half of life are liable. Constant high tension is not only the main cause of premature arterial disease, and consequently of senile decay, but the strain thrown upon the heart by high arterial tension is dangerous, tending to overstrain and degeneration of that organ.

The administration of digitalis and other cardiac remedies must be regulated by the condition of the pulse. Thus in cardiac valvular disease with slow full pulse digitalis should not be given, but only when the pulse is of low tension, *quick or irregular*. If, on the other hand, the pulse becomes irregular during the administration of digitalis, the drug should be at once withdrawn.

It is impossible here to give more than these few hints. The student will learn by constant and repeated observation what important lessons can be learned from the pulse.

## ARTERIAL DISEASE..

§ 67. *SYMPTOMATOLOGY*.—Among the symptoms to which arterial disease may give rise are giddiness or “dizziness,” feelings of faintness, slight syncopal attacks, headache, paroxysmal dyspnoea, gangrene, and other conditions referable to the extremities (Chapter XVII.). But each of these symptoms, excepting the last named, may often be caused by disease of some other physiological system. It is only when several of them are met with together, and an examination of the heart lends confirmation to the idea, that we are led to conclude that the vascular system is at fault. The importance of arterial disease depends more upon its effects on the heart, and indirectly on the other organs and tissues of the body, than upon the vascular condition *per se*.

### § 68. *PHYSICAL SIGNS OF DISEASE OF THE ARTERIES*.—

The physical signs are very few in number, and consist simply of a visible or palpable thickening, dilatation, or tortuosity of the superficial vessels, such as the temporals, radials, brachials, and sometimes carotids. There are three features to note concerning the acces-

sible arteries : (i.) The size of the artery should be observed as we compress, or roll it beneath the fingers—a feature which sometimes requires considerable experience to recognise. (ii.) The thickness of its wall, and it must not be forgotten that high arterial tension may produce the sensation of a thick wall, and *vice versa*. The thickness of the wall is best ascertained by stopping the pulse with the fingers of one hand and rolling the empty tube beyond under the fingers of the other hand. (iii.) By passing the fingers up and down the length of the tube the beadlike thickening due to atheroma may be detected.

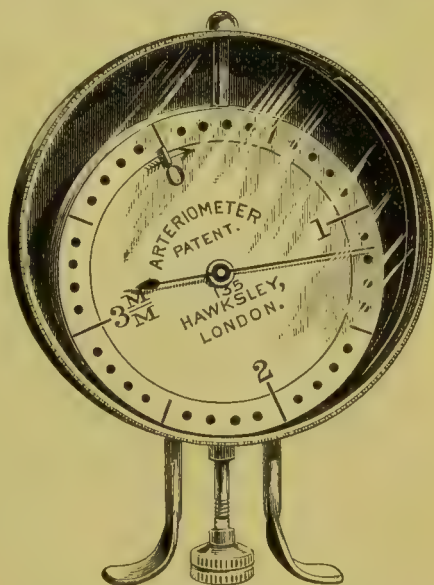


Fig. 32A.—THE ARTERIOMETER is a special instrument which has been designed by Oliver to determine the internal calibre or diameter of the radial artery. See description beneath Fig. 25, p. 128.

The arteries are very much more prone to disease than are the veins, a fact which is in keeping with the greater proneness of disease to attack the left rather than the right side of the heart; at least during extra-uterine life. **The chronic diseases of the arteries** which admit of clinical recognition are as follows:—

I. Atheroma. II. Arterial Sclerosis (Synonyms: Arterial Fibrosis. Arterio-Capillary Fibrosis, etc.). III. Arterial Hypermyotrophy. IV. Functional disease of the arteries. (See Diseases of the Extremities, Chapter XVII.) V. Aneurysmal Dilatation. VI. Chronic Endarteritis, due to syphilis and other causes, is only recognised by its pathological effects (cerebral softening, gangrene, etc., see Diseases of the Nervous System; anatomy). Acute Endarteritis is generally part of Acute Endocarditis (§ 42). VII. Embolism, or the blocking of an artery by an embolus, is the result usually of cardiac disease, especially infective endocarditis; or it may be secondary to thrombosis. VIII. Thrombosis, or the coagulation of blood in a living vessel, is usually the result either of local disease involving the vessel, or of some blood change. Both this and Embolism are dealt with elsewhere. See, for example, Localised dropsy or Phlebitis (Diseases of the Extremities, Chapter XVII.).

§ 69. **Atheroma.** Atheroma has unfortunately come to be used in a somewhat vague sense, but it is taken here to mean a localised or patchy thickening of the tunica intima, occurring for the most part in patients past middle age, unaccompanied, as a rule, by any obvious symptoms during life. It starts as a localised hyperplasia in the deeper (external) layer of the tunica intima; and the change may go on to fatty, caseous, and sometimes calcareous degeneration. When it is advanced the middle and even the external coats may be invaded. It is generally more or less widespread, but the disease nearly always commences and predominates in the larger vessels—*i.e.*, in the aorta and its branches. Consequently, if it be detected in the radial or temporal the inference is that its distribution is extensive, and that it involves the vessels of the brain also.<sup>1</sup>

*Symptoms* are generally altogether wanting.<sup>2</sup> However, when the disease involves the aorta (and it nearly always commences in that situation), it impairs the elasticity of that structure, and gives

<sup>1</sup> Some observers say that, with the exception of the coronary arteries and the vessels of the brain, it does not usually affect the smaller arteries, but I have satisfied myself of its existence in the radials and nearly all the arteries of that size in the body in advanced cases of atheroma.

<sup>2</sup> This absence of symptoms has sometimes led me to conjecture whether atheroma might not be, in a sense, a conservative process, an idea which is further strengthened by two other important circumstances—*viz.*, (1) That the change was most constantly found in those situations exposed to the shock of the systole, as, for instance, on the upper aspect of the arch of the aorta and in the angle of the bifurcation of arteries. (2) It was a constant, and therefore, one might say, a physiological change in greater or less degree in *all* elderly people, dying in the Infirmary, no matter what might be the cause of death. One of the very notable cases was that of a woman aged 100, who died of pneumonia, and who had not at any time presented any cardio-vascular symptoms during life.



rise to an accentuated second sound of the heart in the aortic area. It also imparts to the pulse that feature called by Broadbent "virtual tension" (Figs. 33 and 20, B.), which is a very constant feature of the senile pulse (p. 134). In the more advanced cases atheroma may sometimes be detected as a nodular or beaded thickening in the radial, temporal, and other superficial arteries, which can be felt by moving the fingers up and down the artery. The patient is, however, unaware of its existence, and may, like the case mentioned in the footnote to p. 144, live to old age, unless he be subject to high tension or other cardio-vascular disorder.

*Consequences.* Histologically the change is almost indistinguishable from the syphilitic endarteritis which occurs in younger persons, and it is only by the age of the patient and the history of syphilis that the latter can be differentiated.<sup>1</sup> There is, however, a third im-

portant difference—viz., whereas syphilitic endarteritis commonly results in thrombosis, atheroma rarely does so, unless degenerative changes occur.

Hæmorrhage is apt to occur in these cases,

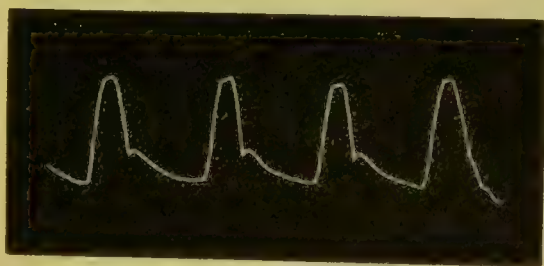


Fig. 33.—TYPICAL SENILE PULSE. W. P., æt. 88. Shows the flat top of virtual tension, which is the chief characteristic of the senile pulse.

due not to the atheroma but to thinning and dilatation of the vessel on the proximal or distal side. Occurring, as it does, only in old people, it is accompanied by, and probably aids in the production of debility and other signs of old age. Probably these are caused by the malnutrition from the loss of elasticity and contractility in the arteries. A case is mentioned elsewhere (Chapter XIX.) in which the debility was so extreme that it was mistaken for paralysis.

No *treatment* will remove the atheromatous condition, but its presence is one of the indications for the avoidance of the causes of high arterial tension.

§ 70. **Arterial Sclerosis.** The term "arterial sclerosis" is here used in a purely clinical sense, as a generic term to mean any

<sup>1</sup> Some (e.g., Heubner) say that they are not identical because fatty and calcareous degeneration do not occur in the syphilitic disease; but this difference is fully accounted for by the difference in age of the patients in whom the two diseases are found.

wide-spread thickening and hardening of the arterial coats which leads to loss of elasticity and contractility of the arteries, which is *clinically recognisable*. It is too often forgotten that what may be called the "parenchyma" of the arterial system, that is to say, its functionally active part, is the middle or muscular coat. Upon the

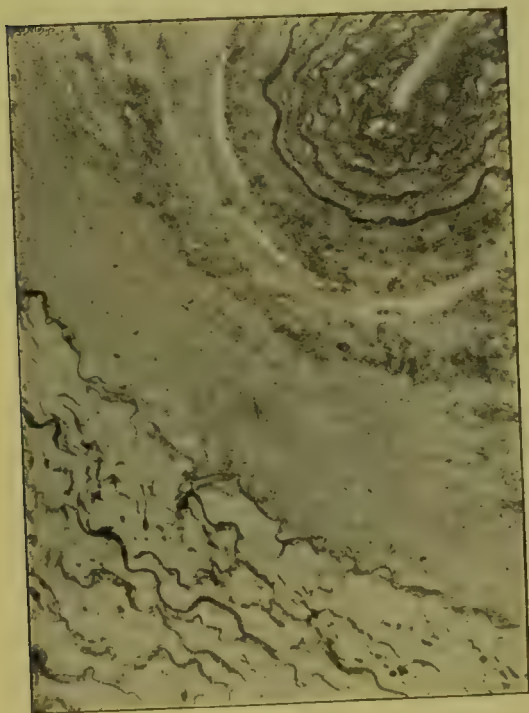


Fig. 34.—ARTERIAL SCLEROSIS. Section Radial Artery magnified about 400 diameters, stained with acid orcein to show granular degeneration of muscle fibres in the mid. coat, which commences and predominates in the *int. layers of mid. coat*. This method of preparation brings out the elastic tissue, as may be seen in tunica advent. and tunica intima. Similar sections stained sufficiently long in logwood brought out the rod-shaped nuclei of the muscle fibres, showing that the change is not a fibrosis, as Gull and Sutton maintained. The appearances described by them as arterio-capillary fibrosis can always be produced by prolonged hardening and by insufficient staining with logwood.

proper functioning of this coat depends the whole of the regulator mechanism of the arterial system. My experience at the Paddington Infirmary, which offered a very extensive field for researches into the pathology of the vascular system, went to show that although very wide changes might exist in the tunica intima or tunica adventitia without symptoms, very slight changes in the media were nearly always attended by some kind of symptom or effect during life. There are certainly several different kinds of histological changes which might come within the

meaning of "arterial sclerosis" used in the above clinical sense.<sup>1</sup>

<sup>1</sup> The subject of arterial disease is surrounded with considerable obscurity, partly because the same words are used by different observers in very different senses, and partly because the several disorders to which the different arterial tunics of the vessels are liable are so ill understood, on account of the difficulty of making a sufficiently exhaustive examination of the entire arterial system in any given case. Ten or a dozen sections of the liver, for instance, will give one a very fair idea of a morbid change in that organ; but to form an accurate conception of the structural changes in the arterial system in any

*Symptoms.* The clinical course of the disease may be divided into two stages: (a) that in which the ventricular hypertrophy is sufficient to compensate for the increased peripheral resistance, and (b) that in which the left ventricle begins to fail—that is, to yield and dilate.

(a) In the first stage, (1) the patient may be free for many years from any symptoms referable to the vascular system, so long as the increased peripheral resistance is adequately and not excessively compensated for by the ventricular hypertrophy. Sometimes the patient may come to us for loss of vigour or breathlessness; but more often the thickened vessels are discovered, so to speak, by accident, when the patient comes under our notice for some other malady. On examination, however, we may find that the arteries are visibly and palpably thickened at the wrist and on the temples, being cord-like, and sometimes elongated and tortuous. The feel of the vessel much resembles that of high arterial tension (a condition, by the way, which may co-exist in the earlier stages), but by compressing the pulse above and so emptying the vessel, and then feeling the artery beyond, the thickening of the wall may be readily revealed. By sliding the fingers up and down it may be distinguished from the beading of atheroma. (2) As the disorder progresses, the patient loses his former vigour, mental and physical. He is, in a word, “old before his time,” for it has been truly said a man is “as old as his arteries.” This loss of vigour is no doubt due to the fact that all the organs and tissues are deprived of that regulation of nutrition which depends upon the elasticity and healthy contractility and relaxation of the muscular tissue of the arteries. (3) Breathlessness, and a tendency to rapidity of pulse after slight, or even without exertion, are, in my experience, the next most constant features. Sometimes the dyspnœa is paroxysmal, and the case resembles asthma. The heart is irritable, and this is evidenced clinically by breathlessness and a pulse which easily becomes rapid and irregular.

(b) When the second stage is reached, symptoms arise which as a whole form a distinct and well-marked clinical picture. They

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given case it may be necessary to carefully examine and measure one or two hundred sections, or more, taken from many different parts, both of the same and different vessels, and to make very accurate measurements of these, both naked eye and microscopic.

are due partly to failure of the arterial functions, but mainly to the failure of cardiac compensation. In addition to the preceding, which become emphasised, the symptoms are as follows: (1) Vertigo may have occurred before the second stage is reached, but the attacks now become more frequent. Indeed, about nine-tenths of the cases of senile vertigo met with in the Infirmary could be traced to this malady. It is not, however, a true vertigo, but rather a sensation of "swimming in the head," or "dizziness," and the patient feels either as if he were "going to fall" or "going to faint." So frequently did these attacks occur among the aged and so-called "healthy" old people in the Workhouse, that they used to pay but little attention to their frequent falls due to this cause. (2) Actual fainting attacks are met with less frequently, but when present they are of more serious import. They vary from a slight interruption of the continuity of thought on the one hand, to a prolonged faint or epileptiform seizure, on the other. (3) Physical signs pointing to a varying degree of hypertrophy and dilatation of the heart are revealed on examination, together with accentuation of the second sound over the aortic cartilage, if high tension be present. (4) Anginoid attacks, or true angina pectoris, is another symptom. Quite three-fourths of the cases of angina pectoris met with in the Infirmary could be traced to this condition. (5) In extreme states of the disease, especially when accompanied by atheroma or endarteritis, there may be gangrene of the extremities, cerebral softening, either localised or diffuse, and similar changes in other viscera. Miliary aneurysms may arise in the periphery of the arteries of the brain, and hæmorrhage in this situation is one of the most frequent consequences. There is always in these cases a tendency to the development of low forms of inflammation, especially "senile pneumonia." (6) Renal fibrosis (chronic Bright's disease—*i.e.*, granular or gouty kidney) is very frequently, but not necessarily, associated with arterial sclerosis. Some hold that chronic Bright's disease is essentially a generalised arterio-renal disease, and that, although arterial thickening may occur without renal disease, the latter is always attended by more or less arterial change.

In the *Etiology*, (1) Heredity is certainly one of the most



important factors, and families are found in which every member shows a tendency to this disease on reaching a certain age. (2) As regards age, the disease is only met with in the latter half of life;<sup>1</sup> and it is rather more frequent among men. (3) The pathology of the complaint is not yet worked out, but in many cases careful inquiry will reveal one or more of the causes of a constantly high tension (*q. v.*). Various toxic blood states possibly act in this way, and there is often a history of alcohol, lead or gout. (4) Overwork and physical strain, by leading to an overfilling of the arteries, are also possible causes, on the principle that increased function causes first increase of structure, and later, degeneration of the muscular coat.

*Diagnosis.* Arterial sclerosis may have to be diagnosed from (1) high arterial tension, by stopping the blood current and examining the artery beyond; (2) atheroma, which gives to the vessel an unequal or beaded character. (3) Granular kidney in its slow insidious onset and vague symptoms closely resembles arterial sclerosis in its clinical history, and can only be distinguished from it by the presence of urinary changes. The arterial and the renal changes are frequently associated. (4) Other causes of progressive debility (Chapter XIX.) may have to be distinguished from arterial sclerosis.

*Prognosis.* In the first stage, though nothing can be done to abolish the thickening of the arterial walls, much may be done to prevent its advance; and if the patient escape pneumonia and other inflammatory conditions to which he is liable, he may live many years. The whole question of prognosis turns very largely on the state of the heart. If the breathlessness is considerable, and the physical signs show marked cardiac dilatation, and the pulse is irregular and rapid, the patient is not likely to live more than a year or two. If, on the other hand, cardiac compensation is good and the patient feels but little distress on movement, then the outlook is not unfavourable.

*Treatment.* The indications for treatment are (a) to keep down the arterial tension, (b) to aid the heart, and (c) to avoid any extra strain being thrown upon the heart or vessels. (1) Hygienic

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<sup>1</sup> Syphilitic arterial disease which occurs in younger subjects is usually more or less localised, and therefore does not come within the scope of our opening definition.

measures are of great value, and the patient's duration of life will a good deal depend upon the kind of existence he can afford to live. He should live a very regular life quite free from any strain on mind or body. The diet should be strictly moderate, and unless heart failure is very pronounced, alcohol should be avoided. The diet should be spare, the amount of proteids being strictly limited, and the digestion carefully attended to. Calcium salts are said to be eliminated by sod. bicarb.  $2\frac{1}{2}$  drachms, neutralised with lactic acid. Six ounces of water are added to this quantity and it is taken in 24 hours. At any rate this relieves many of the symptoms of cardiac dilatation.

(2) The heart needs our special, and often daily attention, and cardiac tonics and other remedies may be administered on general lines (see Cardiac Failure, § 54). The question of Schott baths and passive exercise is a most important one in these cases, and while some advocate them strongly, others say that they are attended with considerable danger by increasing the tendency to hæmorrhage and the other consequences above referred to; but in my belief, if the arterial disease is not very advanced the heart undoubtedly derives considerable benefit by this treatment.

§ 71. **Arterial Hypermyotrophy** is a term which has been employed by the author to imply a generalised increase in the muscular tissue of the arteries. In a paper read before the British Medical Association at Bournemouth, in 1891,<sup>1</sup> based upon a collection of cases which were observed in the Paddington Infirmary, it was shown that a generalised increase in the muscular tissue of the arteries occurred as a distinct clinical and pathological entity, consequent in all probability on states of prolonged or frequently recurring high tension, by the simple physiological law that increased function leads to increased structure. In 1895 Drs. Dickinson and Rolleston<sup>2</sup> showed that a widespread increase of the muscular tissue of the arteries occurs throughout the body in some cases of renal disease. Professor Clifford Allbutt<sup>3</sup> has described under the name Hyperpiesis, a condition which, from a clinical standpoint (for none of the cases were confirmed by autopsy), probably corresponds with the condition which the writer, from an anatomico-clinical standpoint, has called "arterial hypermyotrophy."

The change itself consists of a hypertrophy of the middle or muscular coat of the arterial wall. It affects principally the medium and small-sized arteries of the body, those which normally contain more of this

<sup>1</sup> Other cases of the same condition, in which the muscular tissue also showed commencing degeneration, were published in the *B. M. J.*, January 23rd, 1897.

<sup>2</sup> See *Lancet*, 1895, ii., p. 137.

<sup>3</sup> The Lane Lectures, *Philadelphia Medical Journal*, April, 1900, pp. 400-500; and elsewhere (e.g., *The Hunterian Oration*, circ. 1885).

tissue than is found in the larger vessels. Patients may exhibit no symptoms and rarely die in the early stages of the disease, or until some granular or other degeneration has taken place in the hypertrophied tunica media. The condition, however, is by no means an infrequent one, to judge from the records of the Paddington Infirmary, and if it were not such a laborious task to examine the arteries of the body more cases would doubtless be revealed.<sup>1</sup>

*Symptoms.* (1) The arteries have a thickened but elastic feel, although they may be of normal size. In a few cases the author has been able to confirm this by means of Oliver's Arteriometer. This, the first stage, is usually accompanied by more or less cardiac hypertrophy. It may exist unknown to the patient for many years, and be overlooked by the doctor, or, like arterial sclerosis, discovered accidentally. Sooner or later, however, one or more of these symptoms arise, viz., (2) dyspnœa (sometimes of an asthmatic or paroxysmal character), (3) persistent or recurrent headache, and (4) symptoms of high arterial tension. (5) In the second stage of the disease, when granular degeneration and consequent rigidity are present, the symptoms are indistinguishable from those of arterial sclerosis, which in point of fact supervenes. My youngest case verified by autopsy was 28, but it is more often found in persons over 40.

*Effects.* The results of the thickening in the *first* stage are (i.) a diminution of the lumen of the vessels by reason of the tonic spasm; (ii.) a more or less permanent increase of arterial tension (blood pressure); (iii.) *pari passu* with the arterial thickening and high tension there is hypertrophy of the left ventricle. As the result of the insufficient or ill-regulated blood supply, the tissues are insufficiently nourished, and tend to degenerate, and are more readily prone to inflammation and disease. The patient loses his mental and bodily vigour. In the *second* stage cardiac compensation fails, and the middle coat of the arteries degenerates—the consequences of which are identical with arterial sclerosis above described. Arterial hypermyotrophy is no doubt often associated with granular kidney, probably in about half the cases. But from the cases which the author has collected it is evident that arterial hypermyotrophy may occur without such association.

The *prognosis* of the condition in its early stage is favourable if the patient can live a careful life, although its existence adds to the gravity of intercurrent diseases.

The *treatment* in the early stage is that of high arterial tension (§ 64); in the later stages that of arterial sclerosis (§ 70). Symptomatic treatment is always useful. For the breathlessness, *nux vomica* and *digitalis*; and aperients, especially 1 or 2 gr. of calomel, are at all times useful. For the attacks, especially the vertiginous attacks, nothing

<sup>1</sup> It has been said in criticism of these observations that the change consists of a swelling of the individual muscular fibres rather than a numerical increase (*i.e.*, that it is not a true hypertrophy). This is difficult to refute, because the opportunity does not often occur of examining the arteries before the granular swelling which is also a consequence of the same cause (high tension) has also occurred. But in the first place I would point to the actual occurrence of true hypertrophy in renal cases as shown by Rolleston and Dickinson. Secondly, I have occasionally been fortunate enough to secure cases, dying by accident for example, which undoubtedly exhibited a true hypertrophy without degeneration. Thirdly, a very careful examination of several of my cases shows that there was an actual increase in the unstriated fibres in addition to their degeneration; and, fourthly, Professor Clifford Allbutt's *clinical* observations undoubtedly lend confirmation to the existence of such a pathological condition as arterial hypermyotrophy.

gives so much relief as nitro-glycerine, and for the severe ones occasional inhalations of amyl nitrite. This is an undoubted fact, and constitutes one of the reasons in support of the theory that these seizures are of entirely circulatory origin. It also supports the idea that it is muscular spasm, and not the degenerated arterial wall which produces the vertigo. For the fainting attacks alcohol is indicated in small doses, but I have found that alcohol in anything but very small quantities aggravates the symptoms and consequences of the disorder.

§ 72. **Functional diseases of the arteries.** Of functional diseases, or vaso-motor derangements, we know but little, although several very important maladies are attributed to this cause, *e.g.*, Raynaud's disease and migraine. Functional derangement of the arteries is also manifested by a large number of symptoms, many of which are vague and evident only to the patient. On this account they are apt to be regarded by medical men as unimportant; and it is true that they are not serious in the sense of being lethal, but to the patient they are often extremely disagreeable, irksome, and often terrifying. Of such we may mention alternate flushing and pallor ("flush-storms"), dead hands, cold hands and feet, chilblains, various other erythematous conditions, blue nose, palpitation, tachycardia (§ 43), paroxysms of copious urination, acroparaesthesia, erythromelalgia, feelings of suffocation, pseudo- and true angina pectoris, feelings of tingling, itching, throbbing, and actual swelling of the limbs. A case of the last named is referred to under the heading œdema (Diseases of the Extremities, Chapter XVII.).

**Aneurysmal dilatation of the arteries** belongs to surgery, excepting aneurysm of the thoracic aorta, see § 56, and abdominal aorta, see Chapter XIV. **Embolism** and **Thrombosis** are referred to under Diseases of the Extremities (Chapter XVII.).



## CHAPTER VI.

### THE LUNGS AND PLEURÆ.

OWING to the extreme vascularity of the lungs it is not surprising that inflammation of these organs is a frequent complication of acute general or blood diseases. Thus inflammation of the lungs (pneumonia) is one of the commonest accompaniments of the acute specific fevers, pyæmia, and other microbic disorders. Again, in a generalised blood infection like that which sometimes arises from the tubercle bacillus, the lungs are as we should expect very frequently the seat of tubercular lesions, and there are three *acute* forms of this disease in which the lungs are more or less involved, namely, a tubercular form of acute pneumonia (§ 91a), acute miliary (generalised) tuberculosis (chap. XVIII.), and acute pulmonary tuberculosis (§ 87). Chronic pulmonary tuberculosis (phthisis) is an instance in which the microbe is inhaled; and we meet with the same mode of infection in acute lobar pneumonia and the “air-borne” specific fevers (chap. XVIII.).

#### PART A. SYMPTOMATOLOGY.

The **CARDINAL SYMPTOMS** of diseases of the lungs are **cough**, **breathlessness**, **expectoration** and sometimes **pain in the chest** and **hæmoptysis**. The more general symptoms are pyrexia, emaciation, and debility. The heart, more especially the right side, suffers sooner or later in all serious or prolonged pulmonary diseases which interfere with the pulmonary circulation.

§ 73. Concerning **Cough**, if it is attended by expectoration (as in 1 to 4 below), it points to definite changes either in the lungs or throat. If without expectoration (as in 5 to 8 below), it may point to simple congestion of the throat or larynx, to the presence of pleurisy, to the early stage of some pulmonary disorder, or to some source of reflex irritation. The *causes of Cough* are as follows:—

1. The commonest form of cough is that recurring **WHEEZY** cough attended by expectoration, so typical of bronchitis.

2. **PAROXYSMS** of coughing followed by vomiting occur in *whooping-cough* and advanced *phthisis*. *Bronchiectasis* is attended

by paroxysmal cough with fœtid expectoration at intervals. Paroxysmal cough with or without expectoration occurs with enlarged bronchial glands and other *mediastinal tumours*.

3. The HAWKING cough of throat affections is very characteristic and is met with in catarrhal *pharyngitis*. It also occurs in *nervous* and hysterical subjects. It is also associated with digestive disorders (Chapter IX.), where there is often a collection of mucus in the pharynx, and with chronic liver disease.

4. The IRRITABLE cough, most marked in the early morning and on going to bed, is especially associated with *early phthisis*. There may or may not be much expectoration.

5. A NIGHT cough may be due to a *long uvula*. When a patient complains that a cough is worse at night or on lying down, the uvula should be carefully examined. A slight degree of congestion will cause considerable elongation of the uvula, so that it will irritate the back of the pharynx when the recumbent posture is assumed. A night cough is also associated sometimes with *thread-worms* in children.

6. The long BARKING or nervous cough of *hysteria* is very characteristic. It is unattended with expectoration.

7. The SHORT SUPPRESSED cough associated with *pleurisy* or *pleuro-pneumonia* is so characteristic as to be diagnostic; in the former it is unattended by expectoration.

8. The GANDER or BRASSY cough associated with aneurysm and other mediastinal tumours, is very typical, and when once heard is readily recognised.

9. The REFLEX cough, due to irritation in the area of the pneumogastric, may be caused by (i.) *gastro-intestinal* disorders, such as dyspepsia, constipation, diarrhœa or worms in children; (ii.) *pericarditis*; (iii.) *carious teeth*; and (iv.) *ear troubles*.

The *diagnosis* of these varieties of cough is somewhat important in practice, since they arise from, and may be met with in, affections other than those of the lungs. When a short dry cough is set up by going into the cold, it may be due to pharyngeal congestion or irritation. In simple throat affections the cough comes on in paroxysms, especially after talking. On the other hand, if such a cough comes on in a warm atmosphere, we should suspect phthisis.

The *treatment* of these various coughs depends upon the cause;

but, in general terms, irritable coughs may be soothed by bromides, minute doses of opium, by a linctus of squills and tolu, or by various medicated lozenges.

§ 74. **Breathlessness**, or dyspnœa, is another symptom of lung affections. The causes of breathlessness are dealt with in more detail in the symptomatology of cardiac disorders (§ 21). The types of breathlessness special to respiratory disorders are:—

1. Breathlessness attended by SNIFFING and NASAL BUBBLING is caused by *nasal* or *naso-pharyngeal catarrh*. The obstruction in the nose or mouth usual in such a condition may also cause considerable stertor at night-time.

2. STRIDULOUS respiration, in which the stridor attends both inspiration and expiration, is caused by obstruction in or *pressure* upon the trachea or larynx. It is accompanied, in marked cases, by drawing in of the epigastrium and lower costal cartilages during inspiration.

3. Dyspnœa attended by considerable WHEEZING or rhonchi in the chest is very characteristic of *bronchitis*; attended usually by emphysema.

4. The LABOURED respiration which attends any of the other *gross diseases* of the lungs is different from any of the foregoing. Under this heading also comes the expiratory dyspnœa of *emphysema*, which is due to the fact that the chest remains fixed in a position of inspiration. Undoubtedly the commonest lung condition giving rise to dyspnœa is emphysema, which is revealed by a barrel-shaped chest and hyper-resonance.

5. PAROXYSMAL dyspnœa is sometimes present in asthmatic attacks; but it is more often an indication of *cardiac disorder*, in which circumstances it is often called “cardiac asthma” (§ 21a).

§ 75. **Pain in the chest** is always present with affections of the pleura, but otherwise it is not a constant symptom in pulmonary disorders. The various causes of pain in the chest are enumerated in § 24. The following are the chief types of pain met with in diseases of the lungs:—

(i.) The SHARP, cutting, stitch-like pain of *pleurisy*, before the effusion separates the inflamed surfaces, is greatly aggravated by drawing a long breath. This is undoubtedly the commonest of the pulmonary causes of pain in the chest, and this symptom in *pneumonia* indicates involvement of the pleura. It must be remembered, however, that in some *sub-diaphragmatic diseases*, e.g., of the liver, spleen, or colon, pain is also felt on deep inspiration. (ii.) A SORENESS behind the upper part of the sternum attends the onset of *acute bronchitis*. (iii.) SUDDEN severe pain, followed by considerable pulmonary and general distress, occurs with the onset of

*pneumothorax*. (iv.) SUDDEN pain, attended by hæmoptysis, marks the occurrence of *embolism* of the lung. (v.) *Cancer* of the lung may or may not be accompanied by pain, according to its proximity to the pleura or other sensitive structures. (vi.) All *mediastinal tumours* give rise sooner or later to pain in the chest.

§ 76. **Hæmoptysis** means the spitting of blood [*αἷμα*, blood; *πτύω*, to spit], but the term is confined to the expectoration of blood from the lungs.

The *fallacies* with regard to this symptom are extremely important, and it is sometimes as difficult as it is important to decide whether the blood comes from the throat or nose, from the stomach, or from the lungs. The differentiation is given more fully under hæmatemesis (§ 167a), but it may be mentioned here that blood coming from the lungs is thus characterised:—(i.) It is preceded and accompanied by a tickling cough (if the blood be large in quantity it may excite retching on touching the pharynx); (ii.) the patient usually goes on *coughing* up a little blood for some time afterwards; (iii.) the blood has a bright crimson colour, is alkaline, and aerated (if very profuse it may be dark in colour and unaerated); (iv.) physical signs of disease of the lungs are usually, though not always, present—they may be absent in the early hæmoptysis of phthisis. (v.) The antecedent history of the patient may point to pulmonary tuberculosis, or to cardiac disease, these being undoubtedly the most common causes of hæmoptysis.

*Causes.* For practical purposes the causes of hæmoptysis may be divided into two groups:—

(a) Those which produce slight and sometimes protracted or recurrent bleeding; and (b) Those which produce a copious bleeding at one time.

(a) *Causes of slight and sometimes protracted hæmoptysis:—*

I. PHTHISIS is by far the commonest cause. The hæmoptysis of phthisis may occur either in the early or in the advanced stage of the disease, and in *either* case it may be small or very large in amount. The presence of this cause may be recognised (i.) by the previous and family history of the patient; and (ii.) by evidences of congestion, consolidation, or cavitation of the lung (§ 80). Nevertheless, as just mentioned, the most careful examination may fail to reveal any signs, because hæmoptysis is frequently the earliest symptom of invasion by the tubercle bacillus.



II. CARDIAC DISEASE, especially mitral stenosis or mitral regurgitation, is the next most common cause of hæmoptysis. It may arise in such cases either from congestion, or, more rarely, embolism of the lungs. In both cases evidences of cardiac disease are present. *Congestion* of the lungs is recognised by slightly impaired resonance and the presence of râles at the bases of the lungs. *Pulmonary embolism* (sometimes called pulmonary apoplexy) is known by its sudden advent, and the presence of a cause.<sup>1</sup>

III. VARIOUS PULMONARY DISEASES other than phthisis may be attended by slight hæmoptysis. Thus, in *acute bronchitis*, the sputum may contain streaks of blood from time to time; and in *pneumonia* the sputum is rust-coloured about the third or fourth day of the illness. The hæmoptysis due to *carcinoma* of the lung may be recognised by the irregularity of the physical signs.

IV. ULCERATION of any part of the respiratory passages may give rise to hæmoptysis, small in amount, and apt to be recurrent. A careful examination of the throat and larynx generally reveals this cause.

V. VICARIOUS MENSTRUATION, as a cause of hæmoptysis, is disputed by some. It is recognised by the age and sex of the patient, by its occurrence at the time when menstruation is due, the normal menstrual function being absent, and by the absence of signs of disease in the lungs.

VI. CONSTITUTIONAL or idiopathic causes. Finally, there are certain patients in whom slight hæmoptysis occurs from time to time—the history and examination revealing nothing, and the patient living often to a good old age. The hæmoptysis in such cases is explicable by two hypotheses—the presence of undiscoverable tuberculosis, or a transient congestion due to some

<sup>1</sup> § 76a. **Pulmonary Embolism** is the blocking of one of the branches of the pulmonary artery. It never arises as a primary condition. (a) *Non-septic embolism* occurs most frequently as a complication of mitral stenosis, sometimes also of mitral regurgitation. When there is retardation of the blood in the right or venous side of the heart, it is more apt to occur. Sometimes the embolus consists of a detached portion of a thrombus situated in some part of the venous system (e.g., phlegmasia dolens), but this is not common. (b) *Septic emboli* occur only in connection with septicæmia, and other septic processes.

The *symptoms* of non-septic pulmonary embolism consist of—(1) hæmoptysis, slight in amount, but continuous for a day or two; (2) the sudden occurrence of moderate pyrexia, subsiding in the course of a day or two as suddenly as it came; and (3) a small pneumonic patch can sometimes be made out when it is near the surface. The condition is not usually a serious one in non-septic embolism, and the prognosis is that of the primary condition. But the pulmonary embolism which occasionally complicates the parturient state in the 3rd or 4th week, under conditions which are not yet explained, is speedily fatal. The patient, without any previous warning, simply gasps for breath and dies. The *treatment* of pulmonary embolism resolves itself into that of the primary condition.

constitutional cause. Thus, Sir Andrew Clark found hæmoptysis was of fairly frequent occurrence in subjects of the arthritic diathesis.

(β) *Causes of hæmoptysis in which there is a considerable quantity of blood at one time.*

I. PHTHISIS. Copious bleeding (which may be continuous, perhaps for hours or for a day or two), without ending fatally, is almost invariably due to pulmonary tuberculosis. The chief features by which it is recognised are given above.

II. Rupture of an ANEURYSM into the trachea or bronchus is a by no means rare accident in the history of that malady. It is the one cause of hæmoptysis which is usually followed by immediate death, though in some cases there may be a considerable leakage going on for a day or two before the final issue (§ 56).

III. ULCERATION of the larynx, throat, etc., though usually causing small and recurrent hæmorrhages, occasionally leads to a large amount of hæmorrhage.

IV. Purpura, hæmophilia, scurvy, leucocythæmia, and some other BLOOD CONDITIONS may be attended by bleeding from the lungs. These causes are for the most part rare, but when present are readily recognised.

*Differentiation.* In order to arrive at a diagnosis of the cause of hæmoptysis in any given case, we must *first* of all examine the chest (lungs and heart) very thoroughly; *secondly*, use the laryngoscope to investigate the larynx and naso-pharyngeal passages; and *thirdly*, we must inquire into the patient's history.

The *prognosis* depends, of course, upon the cause. Hæmoptysis is nearly always a serious symptom, and when profuse is followed by considerable debility. In this way it may hasten the end of an advanced case of phthisis. But the hæmoptysis of early phthisis, though indicating definite involvement of the lung tissue, is not so serious, and with proper precautions the patient may completely recover and live to old age.

*Treatment* (refer to causes). (a) For *profuse hæmorrhage* immediate treatment is necessary. The patient must be kept at absolute rest in bed. Ice is usually applied to the chest, but it should not be kept on in one place longer than twenty minutes at a time. The nourishment allowed must be cold. A hypodermic of morphine, or

full doses of opium with acid. sulph. dil., or turpentine internally, are the most efficacious remedial drugs for early administration. Ac. sulph. dil. (m. x) with alum (gr. v) may then be given every twenty minutes. A large dose of ergotin succeeds in some cases. Whitla recommends turpentine vapour in the room. Other drugs recommended are amyl nitrite, which dilates the arteries and so relieves the veins and capillaries, gallic acid, hazeline, or hypodermics of atropin or digitalis. Full doses of ipecacuanha or other nauseants have also been recommended.

(b) When hæmoptysis occurs *in small quantity*, calcium chlor. (gr. xx every four hours) renders the blood more coagulable, and is specially useful. The hæmorrhage of congestion due to cardiac disease should not be checked, unless it tends to become excessive, as it relieves the pulmonary congestion. When hæmoptysis occurs in elderly arthritics, give a sedative cough mixture, saline purgatives, iodides, and cod liver oil.

#### PART B. PHYSICAL EXAMINATION.

The physical examination of the lungs is carried out by means of Inspection and Mensuration, Percussion, Palpation, and Auscultation.

§ 77. **Inspection and Mensuration.** The inspection of the chest must be carried out in a good light, and the patient must be instructed to stand or sit erect and to breathe deeply. After noting the movements from the front, examine the back, then look from above over the clavicles in order to make out the slightest distortions or inequalities of the chest. By inspection and mensuration we note—1, the rate and character of the breathing; 2, the shape and size of the chest; 3, the chest capacity. The chief landmarks of the chest are mentioned in § 31, and the regions into which for descriptive purposes it is divided, are given in Fig. 35.

1. The *rate and character of the breathing* varies normally from 15 to 20 per minute, or one-fourth the rate of the pulse; and whether this proportion of pulse-respiration ratio is maintained should always be observed. Notice whether the breathing is rapid, slow, shallow or irregular. The respiration should be counted without the patient's knowledge, and it is a good plan to

feel the radial artery while counting the breathing, as if you were examining the pulse. Both sides should move equally. Flattening or immobility of any part of the chest points to disuse of that part of the lung, *e.g.*, from consolidation. Flattening or protrusion of the interspaces indicates fluid. Drawing in of the interspaces during inspiration is indicative of some interference with the free entry of air into the lungs (inspiratory dyspnoea),

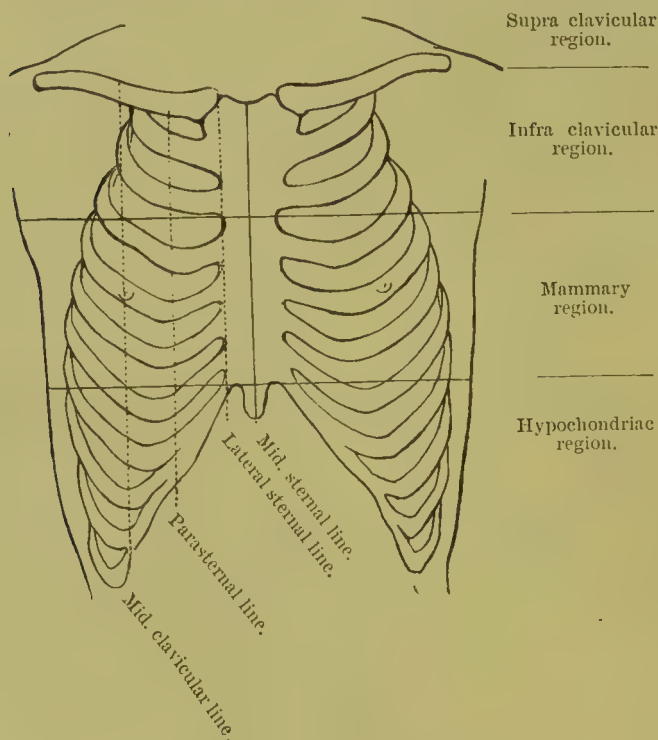


Fig. 35.—Anterior Thoracic Regions.

as in laryngeal diphtheria. *Cheyne-Stokes* breathing is a peculiar rhythmical irregularity of breathing (see § 21).

2. *The shape and size of the chest.* A cross section of the healthy adult chest gives almost the form of an ellipse, the longer diameter being from side to side. In the child it is more circular in shape. The chest should appear symmetrical, although in reality the r. side is slightly larger than the l. There should be no marked hollowing anywhere, the clavicle should form only



a moderate prominence between the supra- and infra- clavicular regions. The circumference of the chest varies with the height of the individual, but it should average for a man 5 feet 6 inches, about 34—35 inches. With deep inspiration it should expand about  $1\frac{1}{2}$  to 2 inches. The measurement at the level of the nipples in the male is a rough measure of the individual chest capacity (see below). The relative shape and capacity of the two sides is measured by a cyrtometer (see also below). The principal abnormalities in shape are the Emphysematous, Phthisical, and Rachitic chests.

The commonest form of abnormality in the shape of the adult chest is the *Emphysematous*, or as it is called, the barrel-shaped chest. Briefly expressed, this alteration consists in the fact that the chest assumes a position of permanent inspiration, and expiration cannot be completely performed. The sternum becomes curved, the lower part being unduly drawn in; and a horizontal section shows the chest to be unduly circular (Fig. 36). When the hands are placed flat upon the chest, on each side, they readily appreciate the fact that in advanced cases there is elevation, but no lateral expansion of the thorax, during inspiration. The upper ribs are unduly crowded together, while the lower ribs are farther apart than normal, and the epigastric angle is very wide. Owing to the permanent elevation of the clavicles and upper part of the chest, and the unusual degree of development of the accessory muscles of inspiration, the neck looks unduly short in an emphysematous subject.

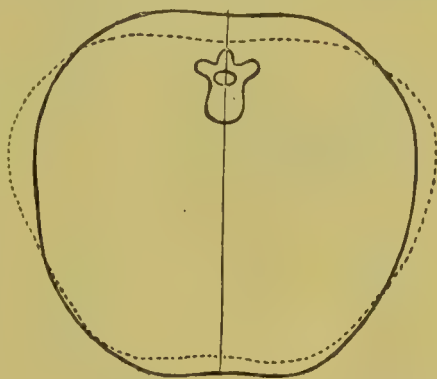


Fig. 36.—CHEST OF EMPHYSEMA. The dotted line represents the normal outline.

The *Phthisical* chest is too long vertically, and in section, too rounded. On inspection from the front it appears flat, but this flatness is more apparent than real, owing to the slipping forward of the scapulæ towards the front of the rounded chest. The form of the phthisical chest corresponds, as Woods Hutchinson

has pointed out,<sup>1</sup> to that of the quadrupeds, and to the undeveloped human chest, or chest of childhood; *i.e.*, the antero-posterior is larger than the transverse diameter.

The *Rachitic* chest is common in children. Owing to the weakness of the bones the chest acquires a characteristic shape (Fig. 37). A vertical groove occurs at the weakest part of the wall of the chest, *i.e.*, down each side of the sternum, just outside the "rickety rosary" or beaded junction of ribs and cartilages (see Rickets, Chapter XVII.). Harrison's sulcus is often present at the same time; it is a horizontal groove at the level of the xiphoid cartilage, running from the middle line in front obliquely outwards and slightly downwards as far as the mid-axilla, along the costal arch.

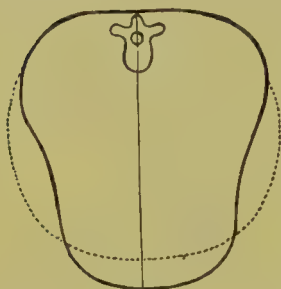


Fig. 37.—RACHITIC CHEST. The dotted line represents the normal outline.

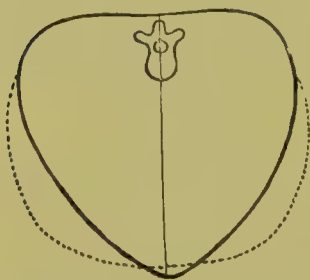


Fig. 38.—PIGEON CHEST. The dotted line represents the normal outline.

The *Pigeon-breast* is found in those who have had some obstruction to respiration in early youth, such as that due to adenoids. The sternum is prominent, the ribs meeting it at more or less of an angle. The cross section of the chest is therefore almost triangular (Fig. 38).

Among the irregular or *asymmetrical abnormalities* in the shape of the chest which the student should look for, are *hollowing, prominence, or contraction.*

(a) *Localised hollowing* or "flattening" of the infraclavicular region may indicate phthisis, or any disease rendering the underlying part of lung useless for respiration.

(β) *Undue prominence* on one side of the ribs anteriorly may be due to:—(i.) Scoliosis, *i.e.*, lateral curvature of the spine, the convexity of the chest being in the opposite direction; (ii.) intrathoracic tumour, fluid, abscess, or air (pneumothorax) in the chest. (iii.) If the cardiac region be prominent it may be the result of cardiac disease in early youth, before the ribs were fully developed,

<sup>1</sup> Woods Hutchinson, *Brit. Med. Journ.*, vol. ii. 1899, p. 1176.

and possibly an adherent pericardium. (iv.) An enlarged liver or spleen may also cause a bulging of the lower-ribs on the r. & l. sides respectively.

(γ) *Contraction* of an *entire side* of the chest which may be due to:—(i.) collapse of a lung (§ 104); (ii.) previous empyema (§ 90); (iii.) chronic interstitial pneumonia and fibroid phthisis (§§ 101 and 98a).

3. *Chest capacity* is used more as a test of health and stamina of the individual than as a sign of pulmonary disease. It is tested either by measuring the circumference of the chest (measurements below), or by the use of the *Spirometer*.

The *Spirometer* is an instrument which registers the “vital capacity,” *i.e.*, the greatest amount of air that can be forcibly expired from the lungs.

TABLE VI.—*Size and Capacity of the Chest.*

ADULTS (AT 30.)				CHILDREN.			
Height.		Average chest measurement. <sup>1</sup>	Chest capacity (Spirometer).	Age.	Average height.		Average chest measurement.
Ft.	Inch.	Inches.	Cubic inches.		Ft.	Inch.	Inches.
5	1	34	175	10	4	5½	25¼
5	2	35.1	177	11	4	7	26¼
5	3	35.7	180	12	4	8¾	27½
5	4	36.2	195½	13	4	10½	28½
5	5	36.8	203½	14	5	0¾	29½
5	6	37.5	214	15	5	3	30¾
5	7	38.1	225½	16	5	5	32½
5	8	38.5	229	17	5	7	34¼
5	9	39.1	238½	18	5	8	35¼
5	10	39.6	246				
5	11	40.2	250½				
6	0	40.8	260½				
over 6 feet		41.0	276				

The *Cyrtometer* is an instrument consisting of two flexible pewter, or pure tin, bands joined by a hinge and graduated in inches. It is used to measure the *relative size* and *shape* of the two sides. Place the hinge *exactly* opposite the spinous processes posteriorly, and bend the pewter limbs round to the front, following the contour of the chest precisely. The instrument is then placed on paper, and the outline thus obtained may be marked by running a pencil round the band. Compare the outlines so obtained with Figs. 36, 37, and 38.

§ 78. **Percussion** is, after inspection, the next step in the examination of the chest. There are two kinds of percussion, *immediate* and *mediate*. In the latter a piece of ivory or wood is placed on the chest, and is struck by a small hammer, or with the finger. The *immediate* is the more usual form of percussion. To elicit the normal resonance of the lungs percussion should be

<sup>1</sup> The mean between deep inspiration and expiration.

stronger than when applied to make out the cardiac dulness. Begin at the apex and percuss *alternate sides* at exactly corresponding points in order to *compare the healthy and unhealthy side*, and thus work gradually downwards. Place the 1st or 2nd finger *firmly* and *flat* against the chest, in a horizontal position, *i.e.*, parallel to the suspected line of dulness. (Only in suspected mediastinal tumour should it be placed vertically.) Then strike upon it with the tips of all the fingers of the right hand. The blow should come from the wrist, not the elbow; and the "staccato" movement should be imitated. Some use one (the middle), two, or three of the fingers of the percussing hand, but this makes the stroke too light, unless, as sometimes happens, it is desirable to demonstrate the delicate shades of pitch, intensity and quality of the sound.<sup>1</sup>

When examining the *back* of the chest (Fig. 39), the patient should be instructed to cross his arms and bend a little forward, so that the scapulæ are drawn out of the way. The normal resonance of the lung note posteriorly extends to the upper border of the eleventh rib on the right side, and the lower border of the eleventh rib on the left side. On deep inspiration the resonance extends an inch lower; and during deep expiration an inch higher. Owing to the thickness of the scapular muscles the note over the scapulæ may be quite dull in muscular people. To examine the *sides* of the chest the patient should be told to put his hands on the top of his head.

The normal pulmonary note can only be learned by practice and experience, and the student should *constantly practise first on normal chests*, so as to accustom himself to the normal resonance; and afterwards on abnormal chests.

The percussion note is normally resonant. It is *dull*, or flat, when the lung tissue is too solid, as in pneumonia; or when the chest contains fluid, as in pleuritic effusion. The percussion note is *hyper-resonant*, or tympanitic, whenever the lung tissue is unduly open, *i.e.*, too full of air, as in emphysema, when there is

<sup>1</sup> The *pitch*, or tone, of the note is its position on the scale, and the more solid the structure the higher the note; the more hollow, the lower and more drumlike or tympanitic. The *intensity* of a note depends on the solidity of the wall of a cavity as compared with its size. The *timbre* or quality of a note is a characteristic which depends on the nature and structure of the vibrating body; just as a wire string and a gut string, though producing the same note, possess a different timbre or quality.



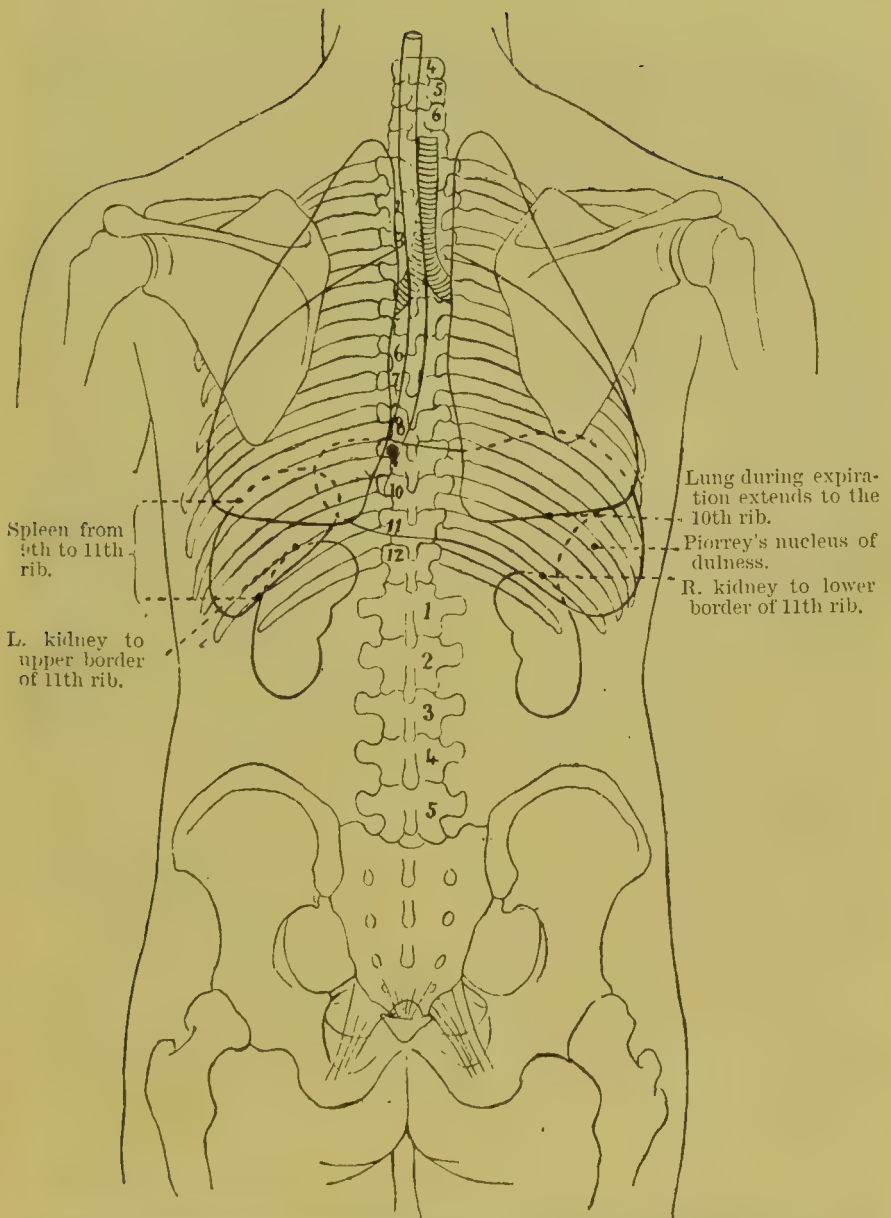


Fig. 39.—THE LUNGS AND OTHER VISCERA FROM THE BACK. The right lung has three lobes, the left lung two only, and the positions of the fissures are shown in the figure. The lines on the figure are only approximate guides; when accuracy is desired the exact position of the lung fissures is obtained by ausculto-percussion (§ 81). A rough guide to the upper border of the lower lobes is found in the position of the vertebral borders of the scapulae when the patient crosses his arms in front of him, and places each hand on the opposite shoulder. The great fissure, which separates the middle and lower lobes, on the r. side, and the upper and lower lobes on the l. side, is indicated on both sides by drawing a line from the 2nd dorsal vertebra to the junction of the 6th c.c. with the sternum. The fissure separating the middle and upper lobes on the r. side is found by drawing a line from the junction of the 4th c.c. with the sternum to meet the line of the great fissure in the mid-axilla.

a cavity near the surface, or when there is air in the pleura (pneumothorax). Cracked-pot sound is a modification due to a large cavity (see Phthisis, § 98). It is normal in children, in whom it is due to the great elasticity of the ribs. When one part of the lung is over-distended with air, as it is in the part which is above a pleuritic effusion (which compresses the lower part of the lung), or above a pneumonic consolidation, the note is unduly resonant. This kind of resonance is called the "Skodaic resonance"; and it may be almost tympanitic (drum-like) in character. It is due to the relaxation of the healthy lung tissue, and the increased amount of air which it contains.

**Gairdner's line.**<sup>1</sup> It is useful to remember that a line drawn from the l. anterior axillary fold to the umbilicus is normally *resonant throughout its entire length*. Abnormally it may be impinged upon anteriorly by consolidation in the upper part of the l. lung, cardiac enlargement, or by enlargement of the liver; and posteriorly by consolidation or fluid at the base of the lung, splenic enlargement or other abdominal tumours.

**Resistance** is another quality which can be observed in the process of percussion as above described. This property of resistance, possessed by solid organs, or the more solid parts of diseased lungs, can perhaps be best elicited by placing a finger of the right hand over an intercostal space and lightly pressing upon the part. It is more applicable to the process of eliciting the boundaries of solid organs, such as the heart (see § 35).<sup>2</sup>

§ 79. **Palpation** is the next step in the routine examination of the lungs. By palpation *Vocal Fremitus* (V.F.), or the vibration of the voice, can be felt. It is scarcely appreciable in women or children with high-pitched voices, but is marked in the adult man. The V.F. is normally greater at the r. than at the l. apex.

This test is of the greatest value in differentiating solid and fluid. Thus the V.F. is *increased* where there is consolidation of the lung, as in pneumonia or phthisis, whereas it is *diminished* or absent when the lung is separated from the chest-wall by fluid or air, or when air is not entering the larger bronchi, as in cases of obstruction of a bronchus. Not only is the V.F. a valuable differential sign, but its degree of diminution is a useful measure of the *amount* of fluid present in cases of pleuritic effusion.

In bronchitis the rhonchi can be felt—*rhonchial fremitus*; and in pleurisy and pericarditis the *friction* can be distinctly felt by the hand.

<sup>1</sup> It was Sir William Gairdner who emphasised the value of this line in physical diagnosis but I am not certain if he was the first to do so.

<sup>2</sup> Robt. Maguire, *Practitioner*, April, 1897, p. 371, may also be consulted.

§ 80. **Auscultation.** In using the single stethoscope place the small end flat against the chest, and, *while it is held in this position* by the finger and thumb, adjust the ear to the other end. The tendency of beginners is to adjust the stethoscope to the ear, but this should be carefully avoided, else the *chest end gets tilted* and the chest sounds are imperfectly conducted. The ear may be placed directly against the chest, but it does not localise the sounds so well. In auscultation there are four things to be observed:—a. The character of the respiratory murmur (R.M.); b. The relative length of inspiration and expiration; c. The presence of adventitious sounds within or outside the lungs; and d. The voice-sounds or vocal resonance (V.R.).

a. The normal CHARACTER OF THE BREATH SOUNDS, *i.e.*, the vesicular or “respiratory murmur” (R.M.), caused by the air entering and leaving the air vesicles, should be listened to in healthy chests as often as possible. It has a soft whiffing character; expiration can hardly be heard, but if heard, there is normally no pause between it and inspiration. The R.M. is normally very loud in children; when a loud R.M. is met with *in adults*, it is called “*puerile* breathing.” The breath sounds are in most persons harsher over the r. apex. The breath sounds are INCREASED, *i.e.*, the breathing is “tubular” or “bronchial”<sup>1</sup> when the lung tissue is unduly solid, as by tubercle or pneumonia, or when a new growth lies between the larger bronchial tubes and the surface. In this condition the sound produced in the larger bronchi is conveyed direct from them to the ear owing to the increased conductivity of the solid lung substance. *Bronchial* breathing can be heard *normally* by listening over the upper segment of the sternum, or near the 4th dorsal vertebra at the back. It has three features—inspiration and expiration are of equal length and character, have an interval between them, and are both rough. *Cavernous* respiration is exaggerated tubular breathing, and is heard when the sound produced in a dilated bronchus or cavity is conveyed in like manner to the surface. *Cavernous* respiration is normally heard over the trachea. *Amphoric* breathing is a sound like air entering

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<sup>1</sup> The terms “bronchial” and “tubular” are mostly taught as synonymous, but some schools (*e.g.*, the Edinburgh) teach that there are three kinds of bronchial breathing—high-pitched or tubular; medium-pitched, or true bronchial breathing; and low-pitched or cavernous breathing.

a bell-jar ; and is heard over pneumothorax or a very large cavity. The breath sounds (R.M.) are DIMINISHED or absent when a layer of fluid (which is a bad conductor of sound), or a thickened pleura intervenes between the lung and the chest-wall ; or when the air does not enter the lung tissue owing to obstruction in a bronchial tube.

b. The RELATIVE LENGTH OF INSPIRATION AND EXPIRATION is approximately as 10 to 12, but the two sounds heard through the stethoscope are as 1 to 2 or 3. *Expiration is prolonged* in any disease which involves a loss of elasticity of the lung tissue, such as emphysema, or tubercle in an early stage, etc.

c. The presence or absence of ADVENTITIOUS SOUNDS has next to be noted. (i.) *Pleuritic friction* is produced by the two inflamed and roughened surfaces of the pleura rubbing together. (ii.) Within the lung various moist and dry sounds may be *added* to the respiratory murmur. Thus the presence of excessive mucus or *moisture* in the large bronchial tubes gives rise to "large or bubbling *râles*" as the air bubbles through the fluid. When the small tubes or air cells are similarly affected "small mucous *râles*" or "crepitations" are heard—which resemble the rustling of tissue paper. If the lining membrane of the large bronchial tubes be thickened and dry, or with only a small quantity of moisture present, "sonorous *rhonchi*" are produced, like the snoring of a person asleep. If the smaller tubes are thickened, "sibilant *rhonchi*" are heard (see Fig. 42). *Rhonchi* are often hard to distinguish from friction sounds, but it may be remembered that whereas friction sounds heard during inspiration and expiration, are separated by a short but distinct interval of silence, *rhonchi* are not so separated, but fade one into the other. *Crepitations* sometimes resemble friction sounds, but are distinguished by being audible only during inspiration.

d. The VOICE SOUNDS, or vocal resonance (V.R.). (i.) When the patient speaks, the vocal resonance is INCREASED (*bronchophony*) if the conductivity of the lung substance is rendered greater by the deposition of solid matter, such as tubercular or pneumonic consolidation. If this be so great that even whispered words and not merely the resonance of the voice are conducted, it is known as "*pectoriloquy*" or "whispering *pectoriloquy*."



The reverberation from the wall of a large smooth-walled cavity may produce the same effect. (ii.) The vocal resonance is DIMINISHED when a layer of fluid or air intervenes between the lung and the chest-wall (*e.g.*, in pleuritic effusion and pneumothorax); or when there is a thickened pleura. Nevertheless in a slight pleuritic effusion the higher tones of the voice sounds are sometimes conducted, especially at the angle of the scapula, and resemble the bleating of a goat (hence called *ægophony*).

Clinically, all the diseases of the lungs may be conveniently divided into those with *dulness* on percussion, those in which the percussion note is *normal*, and those in which it is *hyper-resonant*. Those with dulness may be subdivided into two groups—those in which the dulness is due to solid, and those in which the dulness is due to fluid. The clinical features by means of which solidification of the lung is distinguished from fluid in the chest are so important that they are given in a tabular form.

TABLE VII.—PHYSICAL SIGNS OF

	Consolidation of Lung.	Pleural Effusion.
INSPECTION.	{ Movement im- paired. { May be flattening over the part (if in- fraclav. region).	{ Movement impaired. { May be bulging (of intercostal spaces).
PALPATION.	V.F. INCREASED.	V.F. DIMINISHED or absent.
PERCUSSION.	Resonance impaired.	Absolutely dull over fluid.
AUSCULTATION.	{ BREATHING TU- BULAR. { V.R. INCREASED.	{ R.M. ABSENT OR WEAK. { V.R. DIMINISHED.

The most important features are in small capitals.

§ 81. **Ausculto-percussion**, when employed by experienced observers, enables us to define the boundaries of the heart, or of a mediastinal tumour, with greater accuracy. It is also useful to determine the lobe in which disease is situated. In this method the stethoscope (preferably a binaural) is placed over the middle of a lobe, while one coin is tapped on another, first over another lobe, and then over the same lobe, as that to which the stethoscope is applied. The listening ear recognises the difference of the impact in the two cases. The coins are then placed over the supposed margins of the lobes, and by the slighter but stronger impact conveyed to the ear the division between the lobes can be readily defined. In pneumothorax the pathognomonic "*bell-sound*" is obtained by this method. A method of ausculto-percussion with the aid of a tuning-fork is given at foot of p. 161.

§ 82. **Examination of the Sputum.** Much may be learned from an examination of the sputa. First, as regards its APPEARANCE. In simple pleurisy, though the cough is distressing, expectoration is absent (*i.e.*, the cough is "dry"). If the disease be confined to a moderate catarrhal process of the bronchial tubes (*e.g.*, bronchitis), the sputum is white, clear and frothy ("mucous expectoration"). If the process be more severe and suppurative, or if the lung tissue be breaking down, then pus is present and the sputum is yellowish (muco-purulent). Thin watery sputum is expectorated in large quantity in œdema of the lungs. In phthisis when the lung is breaking down the sputum is often voided in thick spherical purulent masses, like coins, hence called *nummular*. In cases of pulmonary abscess, tubercular cavities, and an empyema bursting into the lung, large quantities of almost *pure pus* are expectorated from time to time. Extremely fœtid expectoration is voided in gangrene of lungs and in bronchiectasis. The latter (bronchiectasis) is distinguished by having large quantities of *putrid sputum*, brought up by paroxysms of violent cough at *one time*; while in the intervals the cough and expectoration are those of bronchitis. The bronchiectatic sputum on standing separates into three layers, the upper clear and frothy, the middle granular with mucus, the lower purulent, with thick "Traube's plugs" (p. 171). The foul odour is due to valerianic and butyric acids. In *pneumonia* the sputum is very characteristic, being (i.) almost airless and extremely viscid, so that the vessel containing it may be inverted, and (ii.) tinged with blood, thus having a pathognomonic "rusty" colour. In severe cases, the sputum becomes thinner, frothy, and dark red, the "prune-juice" sputum. *Casts* of the bronchial tubes, which can be seen by the naked eye (Fig. 48a, p. 198), are expectorated in plastic bronchitis, and occasionally in croupous pneumonia; and shreds of membrane in diphtheria. Hydatid cysts, resembling empty gooseberry skins, are expectorated in that rare condition, hydatid disease of the lungs; or when hydatid of the liver ruptures into them. In town dwellers, and those with dusty occupations, the sputum is dark or even black from the presence of carbonaceous and other particles.

MICROSCOPIC EXAMINATION of the sputum. Various *Bacteria*

(e.g., tubercle, pneumo-coccus, influenza) are found in the sputum in patients suffering from certain microbial disorders. The method of detecting these is described in Chapter XX.

In all destructive diseases of the lung, fragments of pulmonary tissue are present, i.e., epithelial cells and connective tissue. The most characteristic is *elastic tissue*, which indicates unmistakably that the lung is breaking down. Elastic fibres are best revealed by taking a small portion of the sputum and boiling it with liquor potassæ, which breaks up and renders clear all the other elements, but leaves the elastic fibres unattacked. These sink to the bottom of the test tube and may be withdrawn by a pipette (precautions, see urinary deposits) for examination under the microscope. They appear as wavy, highly refractile fibres, of uniform thickness with square-cut ends, and are typically arranged as if surrounding an air cell (Fig. 40). Elastic tissue is found in the mouth after meals, so the mouth and teeth should as a precaution be cleansed before the observation is made; but circularly arranged elastic fibres are quite distinctive of breaking down lung tissue. The method of examining for tubercle is given in Bacteriology (Chapter XX.).



Fig. 40.—Elastic Fibres.



Fig. 41.—Charcot-Leyden Crystals.

The Traube "plugs" of a bronchiectatic sputum (p. 170) are little pellets which contain pus and epithelial cells, with needle-shaped fatty-acid crystals. Sometimes elastic fibres are also present in small amount. They are believed to be pathognomonic of bronchiectasis.

*Curschmann's spirals* are found in the sputum of asthmatic patients. They form pellets the size of sago grains, which can be uncoiled to form a thread about an inch long. Microscopically, they are seen to consist of fine mucous fibrils wound spirally round a central core of mucus. They are probably allied to small bronchial casts (Finlayson). *Charcot-Leyden* crystals (Fig. 41) are colourless pointed octahedral crystals, formerly supposed to be pathognomonic of asthma, but now known to occur in the sputum of bronchitis also.<sup>1</sup>

<sup>1</sup> Charcot-Leyden crystals are composed of phosphate of spermin. Pohl considers spermin to be a decomposition product of nucleo-albumen which normally circulates in the blood. Many diseases (especially nervous) are due to excess of phosphate in the system, which, combined with spermin, may form Charcot-Leyden crystals.

They have also been found in the blood of leukaemia. *Hæmatoidin* crystals are brown or yellow needles, or plates, found in cases of old hæmorrhage from any cause. *Cholesterolin* and *tyrosin* crystals are found occasionally in cases where the sputum has been purulent for a long time. Various *parasites* (actinomycosis, echinococcus, distoma pulmonale, etc.) are sometimes found in the sputum. Sarcinae, and oïdium albicans come usually from the alimentary tract.

PART C. DISEASES OF THE LUNGS AND PLEURÆ;  
THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT.

§ 83. **Classification.** For practical purposes diseases of the lungs and pleuræ, like those of the heart, may be divided into ACUTE and CHRONIC, and each of these may be subdivided into those without alteration of the percussion note (*i.e.*, without dulness on percussion), those with dulness, and those with hyper-resonance.

	Acute.	Chronic.
WITHOUT DULNESS.	I. Acute Bronchitis II. Dry Pleurisy III. Acute Phthisis IV. Whooping Cough	I. Chronic Bronchitis (and Plastic Bronchitis)
WITH DULNESS.	I. Pleurisy with effusion (and Empyema) II. Pneumonia— a. Lobar b. Lobular	Common { I. Chronic Phthisis <sup>1</sup> (and Fibroid Phthisis) II. Hydrothorax III. Pulmonary Congestion (or Œdema) IV. Interstitial Pneumonia V. Thickened Pleura Rare { VI. Cancer and other neoplasms VII. Collapse of the lung. VIII. Syphilitic disease.
HYPER-BESONANCE.	I. Pneumothorax	I. Emphysema

**Paroxysmal.**

I. Asthma.

§ 84. The routine procedure here resembles in principle that of diseases of the heart. First, *What is the patient's leading*

<sup>1</sup> There is no dulness in quite the early stages of some cases.



*symptom?* If suffering from lung disease, his cardinal symptom will be one of those mentioned in section A. Breathlessness and cough are the chief cardinal symptoms.

*Secondly*, follow this up with a few questions to ascertain the *history of his illness*, and especially whether *the disease be acute or chronic*. Other important points are whether the patient has been exposed to a “chill”; and whether there is any “lung disease” in the family. Do not use the word “consumption”; it may frighten your patient unnecessarily.

*Thirdly*, proceed to the PHYSICAL EXAMINATION OF THE LUNGS. The routine method is as follows:—

1. Ascertain whether there is any increased rate or other modification in the breathing, or alteration in the shape of the chest (by *inspection*, and, if necessary, by measurement).

2. Ascertain if there be any dulness or hyper-resonance (by *percussion*).

3. Listen to the breath and voice sounds, directing special attention to any part suspected of disease (by *auscultation*).

4. Test the voice sounds by *palpation*.

5. The sputum should be inspected, and, if necessary, examined microscopically.

The chest should always be stripped, and it is more convenient to examine the patient in a sitting posture, if he be not too ill.

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If the illness developed gradually, and is of some standing, and unattended by marked constitutional disturbance, then turn to **chronic pulmonary disorders** (§ 95, p. 194).

If the illness came on recently and suddenly, accompanied by fever, quickened respiration, coated tongue, and with marked malaise, then the case is one of the **acute pulmonary diseases**, p. 174.

There is one disease of the lungs, **ASTHMA**, which comes on in sudden acute attacks from time to time; it is **chronic**, with **acute exacerbations** (§ 94, p. 192).

**Acute Diseases.** We now proceed to percuss the chest. In all acute diseases special attention should be directed to the lower and back part of the chest, just below the scapulæ. Careful percussion of this region will give us important aid in diagnosis.

TABLE VIII.—DIAGNOSIS OF ACUTE DISEASES OF THE LUNGS AND PLEURÆ.

	Percussion Note.	Auscultation.
I. Acute Bronchitis	Normal	R.M. and V.R. normal; Loud moist râles and dry rhonchi.
II. Dry Pleurisy	Normal	Breath and voice sounds normal; Pleuritic friction.
III. Acute Pulmonary tuberculosis	Normal, or scattered areas of dulness.	Scattered fine moist râles may be the only auscultatory signs.
IV. Pleurisy, with effusion	Dull	R.M., V.R., and V.F. diminished; Pleuritic friction at early and late stage.
V. Croupous Pneumonia	Dull	V.R. and V.F. increased; Bronchial breathing; Fine or coarse (redux) crepitations.

The acute diseases without alteration in the percussion note, *i.e.*, **without dulness**—excluding WHOOPING-COUGH, which is an infective disorder, and has no physical signs in the lungs, and ASTHMA, which is of a paroxysmal character—are: I. ACUTE BRONCHITIS; II. DRY PLEURISY; and III. One form of ACUTE PULMONARY TUBERCULOSIS (Acute Phthisis).<sup>1</sup>

I. *The patient complains of a cough, with frothy expectoration, and his temperature is slightly elevated; there is no alteration in the percussion note, but on auscultating the chest, loud RHONCHI are heard—the disease is ACUTE BRONCHITIS.*

§ 85. **Acute Bronchitis**, or inflammation of the bronchial tubes, is certainly the most common disease of the lungs in this climate.

*Symptoms.* The disease commences gradually in the course of one or two days, with a feeling of tightness of the chest, of soreness behind the sternum, shortness of breath, frequent cough, and slight rise of temperature, 100° to 101°. The inflammatory process lasts from ten days to three weeks, and gradually subsides. The sputum is viscid and scanty during the first few days, and then becomes thinner and more easily coughed up.

*Physical Signs.* The percussion note is unaltered unless, as so

<sup>1</sup> In the early phase of this malady there is no alteration of the percussion note, but as the disease progresses a patchy dulness appears, if the patient live long enough.

frequently happens, emphysema be present also, in which case the chest is unduly resonant. On auscultation the vesicular murmur is obscured over the whole chest on both sides by loud rhonchi and

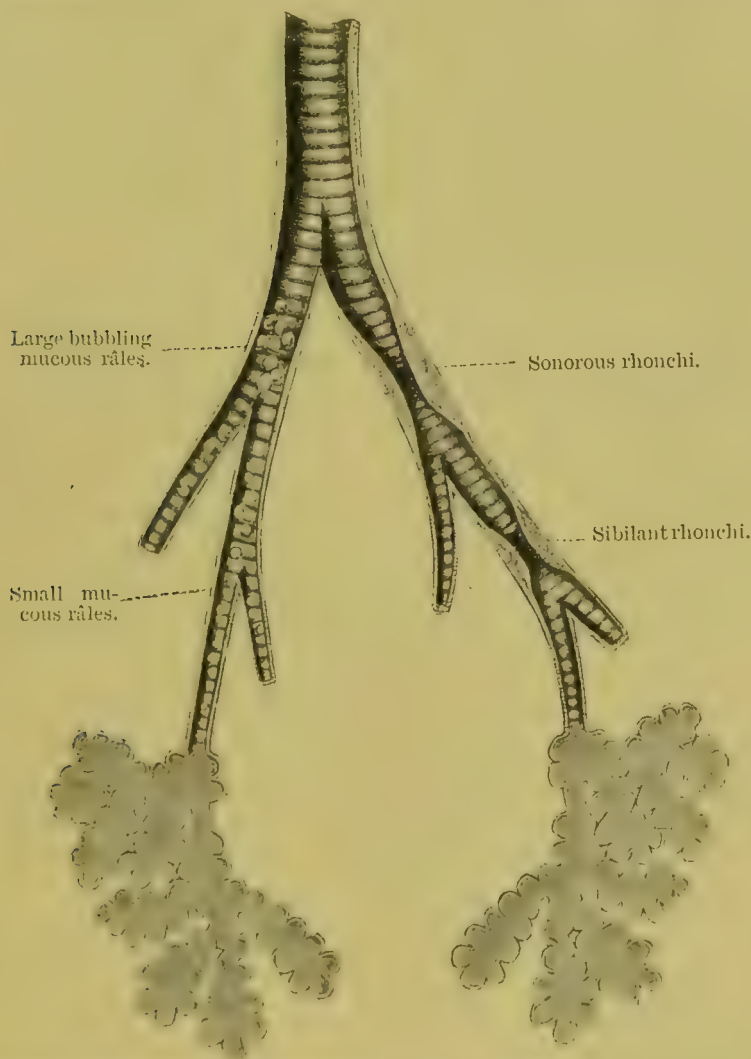


Fig. 42.—Diagram to show the production of *râles* (moist sounds), on left, by mucus in bronchial tubes, and *rhonchi* (dry sounds), on right, by narrowing of the tubes.

moist râles (see Fig. 42). On palpation rhonchial fremitus can also frequently be felt.

*Causes.* Bronchitis is generally attributed to:—(i.) A chill; that is to say, sudden exposure to cold, with a determination of blood to the interior. (ii.) Sometimes, however, it is caused by spreading

from laryngitis. (iii.) It is a frequent complication of many of the specific fevers, especially measles, whooping-cough and typhoid. It is so frequently present with the first and last as to constitute an aid to the diagnosis of those diseases. (iv.) Certain occupations which expose people to irritating vapours and small particles of dust predispose to acute bronchitis. Thus the cotton-mill hands and chemical manufacturers frequently suffer from bronchitis. It is also common amongst cabmen, mariners, and others who are exposed to all weathers. (v.) It is a common accompaniment of many other pulmonary diseases, though it may be a subordinate feature ; and (vi.) it is commonest in childhood and old age.

The *diagnosis* is not difficult in most cases, but *acute tuberculosis* is at first very apt to be regarded as acute bronchitis. The diagnosis is aided by the greater elevation, and the intermitting character of the pyrexia in the former, and by the presence of the tubercle bacillus in the sputum. The *capillary bronchitis* of children is really a *broncho-pneumonia* (q. v.) ; the constitutional symptoms are much more marked, there may or may not be some dulness, and the differentiation from simple acute bronchitis is not always easy.

The *prognosis* is favourable in adolescence and adult life, and it always clears up in one to three or four weeks, though it has a special liability to return, and ultimately to become chronic. It is dangerous in infancy and old age, where the resisting powers are feeble. It is one of the commonest causes of death in the latter. If an attack of acute bronchitis does not begin to clear up in two or three weeks, pulmonary tuberculosis should be suspected, especially if the patient be young.

*Treatment.* The indications are :—(i.) During the first stage, to promote the secretion ; (ii.) when the secretion is free, to stimulate the bronchial mucous membrane ; (iii.) during convalescence, to treat the constitution so as to enable the patient to throw off his liability to bronchitic attacks. At the onset give an aperient and a diaphoretic mixture, with perhaps a few grains of Dover's powder to soothe the pain. Poulticing is useful, and emetics are given to children. To promote the flow of secretion warm alkaline drinks and expectorants such as ipecacuanha and antimony, together with liq. amon. acet., are especially useful. When the secretion is free, that is, after three or four days, stop the antimony, and administer



expectorants such as am. carb., syr. tolu, senega, and squills (F. 57). If the patient is of a gouty or rheumatic diathesis, or the sputum is very tenacious, add pot. iod. to the expectorant mixtures. During the stage of recovery tonics and cod-liver oil are called for. The patient must be confined to bed, and will derive great benefit from the inhalation of steam. In childhood this is best done by a bed canopy and a steam kettle beside it; in adults, a kettle on the fire with a long spout will suffice. Linseed-meal poultices, a turpentine stupe to the chest, or a covering of cotton wool give great relief to the distressing tightness of the chest. See also F. 30 and 68.

II. *The patient complains of sharp PAIN in the chest on inspiration; he has a short dry cough, and his temperature is moderately elevated. On auscultation FRICTION is heard—the disease is DRY PLEURISY.*

§ 86. **Dry Pleurisy** is inflammation of the pleura without effusion. In this disease there is a fibrinous exudation on the visceral and parietal layers of the pleura and a tendency to the formation of adhesions; sometimes to the effusion of fluid.

*Symptoms.* The disease in some cases comes on quite suddenly with a stitch-like pain in the chest. The constitutional disturbance is never very great and the patient rarely takes to bed. The temperature may rise to 100° or 101°, rarely higher. The most marked symptom in this disease is pain in the chest, affecting in most cases one side only, and characterised by being greatly increased on deep inspiration. The pain is caused by the contact of the inflamed pleural surfaces, and is usually, though not necessarily, located over the diseased part.

*Physical Signs.* Percussion reveals nothing. On auscultation the respiratory murmur may be found to be normal, or shortened, as the patient endeavours to restrain the movements of the chest on account of the pain so caused. From the very outset a pleuritic rub is heard—more or less all over one side, but most marked at the angle of the scapula (compare § 80). Sometimes the inflammation undergoes resolution or adhesion, sometimes it goes on to effusion. As effusion takes place, the pain and pleuritic friction disappear, to reappear again when this subsides (as in pericarditis).

*Causes.* (i.) Sometimes it is a primary malady, attributed to

chill, especially in persons of a gouty or rheumatic diathesis. (ii.) It may occur as a complication of some constitutional malady, such as measles or scarlatina. (iii.) Inflammation may extend from disease of the underlying lung, such as pneumonia, tuberculosis, cancer and embolism, or from adjacent organs, such as the liver or spleen. (iv.) Undoubtedly a large number of apparently simple pleurisies are tubercular in origin (some go so far as to say 82 per cent.); and this fact should always be remembered (see also p. 204, and footnote).

The *diagnosis* from *muscular rheumatism* (pleurodynia) is made by the tenderness and absence of friction sound in the latter. *Intercostal neuralgia* has tender points along the course of the nerve, and is not aggravated by deep inspiration. Pleuritic friction is distinguished from the rhonchi heard in *bronchitis* by there being in nearly every case of pleurisy a distinct interval between the inspiratory and the expiratory rub.

*Prognosis.* It is not a serious malady and readily yields to treatment; but sometimes effusion occurs which is apt to become chronic (*vide* Pleuritic effusion). Sometimes this effusion becomes purulent (Empyema, § 90), and the prognosis at once becomes grave.

*Treatment.* Considerable relief is derived by simply strapping the affected side of the chest so as to limit the costal movements of respiration. This may be combined with some local application; that which gives greatest relief is undoubtedly a linseed-meal poultice. As the disease becomes chronic, counter-irritants are called for, more especially iodine, which may be painted on daily until the skin becomes sore. If it does not disappear in the course of a few weeks, we must suspect some other cause for the mischief, such as those mentioned under pleurisy with effusion. Diuretics, diaphoretics, iron, and other tonics are useful.

III. *The patient exhibits the signs of subacute bronchitis; but he has a* HECTIC TEMPERATURE, *and the sputum contains* TUBERCLE BACILLI—*the disease is* ACUTE PULMONARY TUBERCULOSIS.

§87. **Acute Pulmonary Tuberculosis** (acute phthisis, galloping consumption) is a catarrhal process affecting the entire lung tissue, due to the invasion of the tubercle bacillus. It is often part of a tuberculous process infecting the whole body, and is therefore sometimes described as the pulmonary form of acute general tuberculosis (see Chapter XVIII, where a chart is given showing the typical course of the temperature in both diseases).

*Symptoms.* The malady is of most insidious onset with progressive weakness and emaciation. Some weeks before any physical signs are evident the thermometer shows the typical intermittent pyrexia so characteristic of tubercle—an evening elevation of  $101^{\circ}$  to  $103^{\circ}$ , and a morning normal temperature; in some cases the inverse type is present, where the temperature is higher in the morning than in the evening. Night-sweats and cough are present, with muco-purulent expectoration. Dyspnoea and sometimes cyanosis develop, out of proportion to the physical signs. Great weakness ensues, and in the third or fourth week the patient may develop the symptoms of the typhoid state.

The *physical signs* referable to the lungs are most indefinite, or resembling at first those of bronchitis. At first there is no alteration in the percussion note, but by-and-by careful percussion discovers scattered patches of dulness. Auscultation at first may give little help, but in the course of a week or so it reveals rhonchi and fine râles over certain areas which do not shift from place to place as in bronchitis. Later on the râles are coarse and bubbling, and areas of tubular breathing may be found.

The *diagnosis* in the first stage from bronchitis and broncho-pneumonia is extremely difficult. We have to rely upon the disproportionate emaciation and cyanosis, the character of the temperature, and the patchy distribution of the physical signs in tuberculosis. In other cases the malady is almost indistinguishable from enteric fever, excepting for the marked predominance of the pulmonary signs and the absence of abdominal symptoms. In all stages the detection of the tubercle bacillus in the sputum is a valuable aid to diagnosis, though its absence does not exclude acute pulmonary tuberculosis.

*Causes.* The disease may occur at any age, but is commonest in young adults, and in those with a family history of consumption. In some instances, acute general tuberculosis originates from a primary focus, such as a tuberculous joint which had been considered cured. Sometimes the disease follows measles or whooping-cough in children.

*Prognosis.* The disease is almost uniformly fatal in about two to twelve weeks, for we have no means to cope with such widespread disease.

*Treatment* is almost entirely symptomatic. The administration of oxygen and purified air might perhaps be worth trying; and quinine may delay the infective process.

§ 88. *The patient, a child, has PAROXYSMS of coughing which frequently terminate in VOMITING. There is very slight feverishness, but no signs in the lungs, or perhaps a little bronchial-catarrh—the disease is WHOOPING COUGH.*

**Whooping Cough** (Pertussis) is an acute infectious disease, and it will be described among the microbic disorders (Chapter XVIII.).

We now turn to the **acute diseases with dulness on percussion**—I. PLEURISY WITH EFFUSION (Serous or Purulent), II. PNEUMONIA, and III. BRONCHIO-PNEUMONIA.

I. *The patient has a DRY COUGH with moderate fever and other*

*constitutional symptoms.* On examining the chest the *R.M.*, *V.R.*, and *V.F.* are found to be diminished or absent—the disease is PLEURISY WITH EFFUSION.

§ 89. **Acute Pleurisy with effusion.**—When describing acute dry pleurisy (§ 86) it was pointed out that the disease may undergo resolution or result in adhesions. It may also go on to effusion—Pleurisy with effusion.

*Symptoms.*

There is usually a history of a more or less acute onset with pain in the side (§ 86), but as the disease progresses, and the surfaces of the pleura are separated by fluid, pain becomes less and less marked. The patient suffers from general malaise, and finds it difficult to lie on the sound side because the weight of the fluid on the other side impedes the

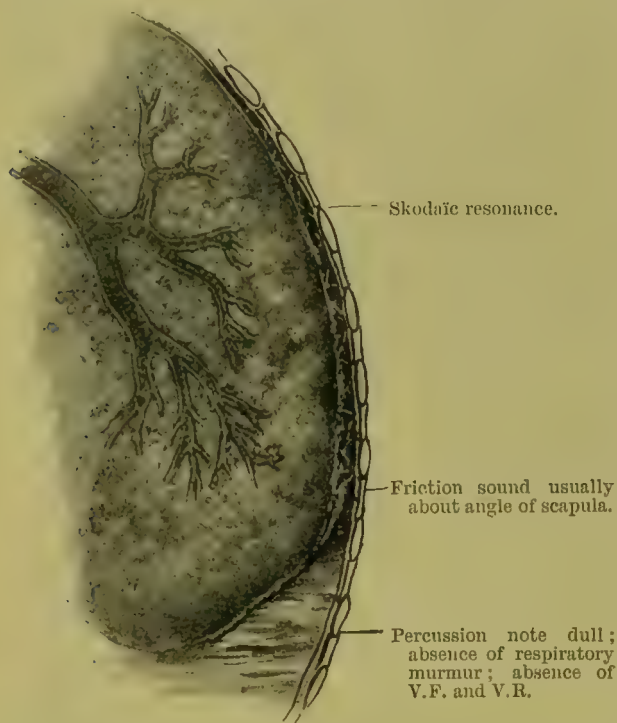


Fig. 43.—Diagram to show physical signs produced in different positions in ACUTE PLEURISY WITH EFFUSION.

action of the lung. A degree of breathlessness may be present, but even with a large amount of fluid this is not invariably a prominent feature.

*Physical signs* (see Fig. 43). Percussion reveals absolute dullness over the fluid. Above the level of the fluid, if the lung be otherwise healthy, there is a hyper-resonant note (Skodaic resonance). When the effusion is large it causes displacement of organs, which may be very considerable (see Fig. 44). The level



of the fluid does not usually shift with the position of the patient, as it does when there is non-inflammatory (dropsical) fluid in the chest. On auscultation over the fluid the breath sounds are absent; the V.R. is greatly impaired or lost.<sup>1</sup> At the upper margin of the fluid posteriorly—say, just about the angle of the scapula—only the highest pitched tones of the voice are transmitted, and they produce therefore a sound like the bleating of a goat (*ægo-phony*). On palpation the V.F. is found to be diminished or absent over the fluid, and there may be bulging of the inter-costal spaces. The amount of fluid present may be estimated by (i.) the degree of diminution of the V.R. and V.F., and (ii.) the amount of displacement of organs. The diagnosis of pleurisy in its earlier stages is referred to under dry pleurisy. The differentiation of the physical signs of fluid in the chest, as compared with those of consolidation of the lung, is so important that it is given in a tabular form in § 80. It is sometimes difficult to make out the left margin of the cardiac area, when there is effusion in the left pleura. Dr. S. H. Habershon has suggested a very valuable

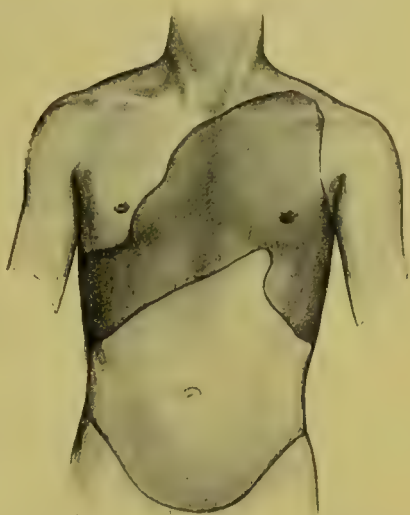


Fig. 44.—Case of PLEURITIC EFFUSION showing displacement of organs (heart and liver). The patient was a boy, *æt.* 12, admitted under the care of Sir Wm. Gairdner, in the Western Infirmary, Glasgow, April 20th, 1895.

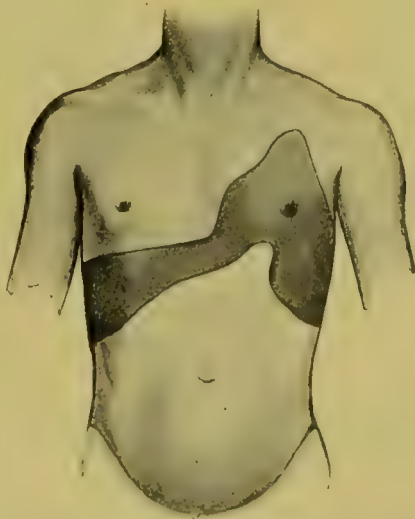


Fig. 45.—Shows altered state of dulness under use of diuretics (April 29th). *Lin.* Hydrarg. was applied, and he took internally *Pot. cit.* ℥ 10, *Pot. iod.* gr. 5, *Liq. Am. Acet.* ℥ 20, *Decoct. Scopar.* ʒii, 4tis horis. Patient also had diarrhœa at this time.

<sup>1</sup> Bronchial or tubular breathing occurs wherever there is moderate effusion, because the lung collapses with the progress of the effusion.

aid in such cases. Place a vibrating tuning fork, such as aural surgeons use, in mid-axilla over the 7th rib. Listen with the stethoscope over the centre of the cardiac area, and gradually move it towards the tuning fork, and in other directions. As the stethoscope crosses the boundary of the heart, there is a distinct difference in the note heard through the stethoscope, and in this way the cardiac boundary may be determined.

*Course and Prognosis.* In about a fortnight from the date of onset the fluid usually shows signs of diminution in quantity, the V.F. and V.R. return, and the breath sounds become more audible. This is the usual course, but several untoward results may ensue:—(i.) the effusion may remain for an indefinite time, and re-collect after tapping. (ii.) Adhesions may take place between the two layers, and considerable thickening of the pleura result. (iii.) The fluid—especially in children after scarlatina—may become purulent (Empyema, see below).

*Treatment.* To get rid of the effusion purgatives, diuretics, and diaphoretics (pot. cit. and bitart. pot. nit., liq. ammon. acet., etc., F. 55) are often efficacious. Iron and other tonics are useful. If these measures fail after a few weeks' trial paracentesis should be performed. (Figs. 44 and 45.) It should be remembered, in recurrent effusion, that tubercle may be in operation.

*Paracentesis thoracis.* The instrument used is an adaptation of the familiar trochar and cannula. We are here dealing with a cavity whose contents are under a minus pressure, so it is necessary to have a pump or exhausted bottle communicating with the trochar (see Fig. 46). The site of puncture—usually the seventh interspace in the posterior axillary line—should be thoroughly cleansed with soap and water, then with turpentine, and a carbolic fomentation 1 per cent. applied for several hours before puncture. The needle of the aspirator should be boiled. The bottle or chamber of the syringe is next exhausted of air. If the point of the instrument be not very sharp, it is desirable to make a nick with a scalpel in the skin, previously pulled downwards over the rib below. Then the instrument is thrust into the intercostal space boldly at the acme of an inspiration. Communication is then established with the bottle or syringe, the flow being regulated by the tap or piston so that the outflow may not be too rapid. Much coughing by the patient indicates that the point is touching the lung. A quantity varying between 5 and 50 ounces may be withdrawn, but the operation must be stopped if coughing or respiratory distress is caused. To seal the opening, all that is necessary is to take a piece of gauze, pour upon it some collodion, apply it to the skin alongside the cannula, withdraw the latter, at the same time pressing the gauze over the site of puncture, and finally fixing it there with adhesive plaster. If the fluid contain blood it may indicate slight wound

of the lung or carcinoma, or occasionally tubercle. If it be purulent the surgical measures for empyema are applicable, and it is wise to be prepared for this eventuality.

Ia. *The physical signs are those of pleurisy with effusion, but it does not clear up in due course, and the patient has SWEATINGS, SHIVERINGS, and IRREGULAR ELEVATIONS of temperature—the disease is probably EMPYEMA.*

§ 90. **Empyema** is a collection of purulent or sero-purulent fluid within the pleura. It most often follows a serous effusion, but it may be purulent from the beginning.

The *symptoms and physical signs* are similar to those of serous effusion (*q. v. supra*), with certain others in addition, viz. :

(1) It may be found that the fluid *does not clear up* as a serous effusion should do, and thus the presence of pus may be suspected. (2) Whenever pus forms, either in the pleura or elsewhere, it is marked by the occurrence of sweatings, shiverings, and an intermittent pyrexia. (3) The aspiration of a few drops of the fluid with a hypodermic needle will often settle the diagnosis, though there are two fallacies in this

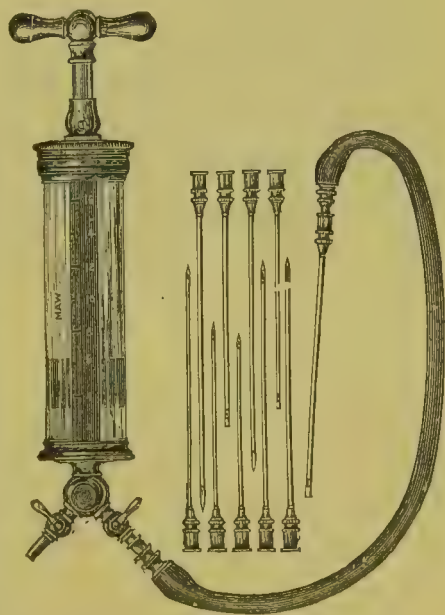


Fig. 46.—Aspirator for pleuritic effusion.

method ; first, in rare cases the fluid may be too thick to come through the needle, or again, the pus may be encysted between the lobes of the lung. In either case, the use of the longer cannula of an aspirator will generally obviate the difficulty. (4) (Edema of the integument, the pointing of an abscess in an intercostal space, over the clavicle, or even in the groin, or copious discharge of pus by the mouth are in rare instances the first distinct evidence of a localised empyema. (5) The history generally throws considerable light on the case by revealing one of the *causes* of empyema, namely :—

(i.) It is far more frequent in childhood than in adults. The

empyema of children sometimes differs from that of adults in the extreme rapidity with which the pus forms, and the frequent absence of the constitutional symptoms mentioned above.<sup>1</sup>

(ii.) Tubercular disease is a very common cause. (iii.) It frequently follows one of the acute specific fevers, especially scarlatina, measles, and small-pox. (iv.) A cavity in the lung may rupture into the pleura. If it is shut off from the bronchi, empyema ensues; if not, pyo-pneumothorax is caused. (v.) The careless performance of paracentesis in a serous pleuritic effusion may lead to empyema. (vi.) Abscesses may burst into the pleura from the spine, the liver, etc.

*Prognosis.* Empyema is always serious, and runs a somewhat prolonged course of some months. Its course can be considerably modified by prompt and adequate surgical treatment. If left to itself the results vary—sometimes there is compression and destruction of the lung; sometimes there is a falling-in of the side of the chest; sometimes, as above mentioned, the pus burrows in various directions; or it may become partially absorbed, and result in a caseous mass.

*Treatment.* Once we are sure the fluid is purulent, aspiration should be immediately performed (*vide supra*). If it re-collects, as it very generally does, a free opening should be made, and one or more ribs resected. Some recommend that free drainage should be proceeded to without preliminary aspiration, on account of the frequency with which the latter fails. In the case of *tuberculous* empyema some advise non-interference. It is certainly a very fatal form. The question is a surgical one. Professor Macewen (of Glasgow) has resected a portion of the lung with success.

II. *The patient has been TAKEN ILL SUDDENLY, the temperature is high, the dyspnœa considerable, and the expectoration soon becomes rusty. There are SIGNS OF CONSOLIDATION at the base of one lung—the disease is ACUTE LOBAR PNEUMONIA.*

§ 91. **Pneumonia**, *i.e.*, inflammation of the pulmonary tissue proper, or parenchymatous inflammation, occurs in two forms. The *first* and most acute is, from its area of distribution, termed Lobar Pneumonia; or, from the nature of the inflammation, Croupous Pneumonia. The *second* is termed Lobular Pneumonia, because it

<sup>1</sup> Many of the remarks relative to Pyo-pericarditis (§ 41a) apply here.



affects the lobules of the lungs (also called Broncho-Pneumonia, Catarrhal Pneumonia, see below).

**Acute Lobar Pneumonia**<sup>1</sup> commences in typical cases very suddenly, with well-marked constitutional symptoms, such as headache, backache, rigor, and in children vomiting. The temperature during the rigor rises to 103° or 104°, and it remains at this point for about a week (Fig. 47). The aspect of a pneumonia patient is very characteristic (§ 7)—the face is flushed, and herpes often appears on one side of the mouth. There is pain in the affected side, short cough, shallow rapid breathing, and on the third or fourth day tenacious rusty-coloured sputum. The pulse-respiration ratio is two to one instead of the normal four to one. The urine is scanty, high coloured, with diminution of the chlorides. The

patient shows more and more distress, and in a short time there is generally delirium, with signs pointing to failure of the heart. About the *seventh* or *eighth* day the fever, in favourable cases, terminates by crisis, falling to normal in the course of a few hours.<sup>2</sup>

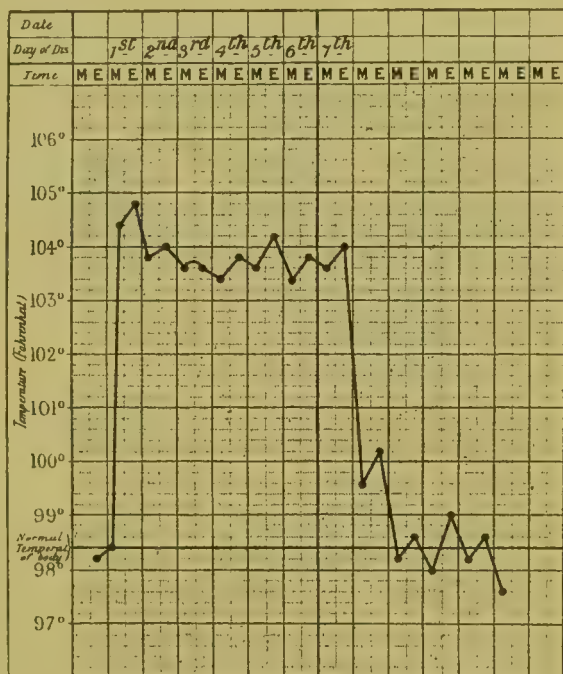


Fig. 47.—ACUTE LOBAR PNEUMONIA, showing typical crisis on the 7th day; Geo. H., æt. 35, was taken ill very suddenly when in the infirmary with shivering and acute pain in the side.

<sup>1</sup> Acute lobar pneumonia was for a long time regarded as the best illustration of a purely local inflammation due to some local cause, such as an injury or a "chill"; but it is now known to be due to the pneumococcus (see Chapters XVIII. and XX.), which appears to have a special proclivity for the pulmonary tissue, because here we find its chief and most constant local lesion. It often attacks those weakened by some other disease. Life is terminated by pneumonia in a large number of chronic and wasting disorders. It is also a frequent and fatal complication of the other microbial disorders.

<sup>2</sup> Crisis often occurs on the odd days, i.e., fifth, seventh, ninth, or eleventh of the disease. Pseudo-crises occasionally occur, but these are distinguished from true crises by the fact that the pulse and respiration do not return to normal. In rare cases the temperature falls by lysis.

This is accompanied by marked general improvement, the pulse-respiration ratio returns to normal, and a critical sweating or diarrhœa may occur. The whole illness lasts about two to three weeks. If it lasts longer, *tuberculosis may be suspected* (§ 91a).

The *physical signs* are limited to one lobe of the lung, usually the lower lobe. It is only in rare cases that both lungs are affected. Percussion may, for the first day or two, reveal no dulness, but, as a rule, there is elicited early in the disease slight impairment of the percussion note, which soon becomes absolute dulness. On auscultation the R.M. is weak, and fine rustling crepitations are heard, which have been compared to the rustling of hair or tissue paper against the ears. As the inflammatory exudation increases the lung tissue becomes solid, and over the dull area we get all the *signs of consolidation* (p. 169). When the fever abates coarse moist râles (reduced crepitations) are heard, and the percussion resonance and normal R.M. gradually return.

*Etiology.* Pneumonia occurs at all ages, and in both sexes, but is commonest in adult males. Debilitating influences, such as exposure, are said to predispose to the disease; but it is surprising how often strong, apparently healthy men are attacked, and these not infrequently succumb. Like other local inflammatory diseases, it may arise as a complication of a constitutional malady; the acute specific fevers in especial rendering a person vulnerable to the pneumococcus microbe. When pneumonia runs an atypical course we should always bear in mind the possibility of the lung affection being only a complication of a constitutional disease such as typhoid fever. Acute lobar pneumonia is itself a microbic disease, the specific cause being a diplococcus, the pneumococcus of Fraenkel.

*Diagnosis.* Acute *pleurisy with effusion* is diagnosed from pneumonia by means of the facts given in the table of diagnosis between consolidation of the lungs and fluid in the pleura (p. 169). *Broncho-pneumonia* runs a different course, and the signs are scattered over both lungs (see table below). The sudden onset of acute pneumonia resembles that of *Scarlet Fever*, *Erysipelas*, and *Small-pox*, but the absence of rusty sputum and altered pulse-respiration ratio distinguishes them. There is a pneumonic

form of *Acute Pulmonary Tuberculosis* which has to be borne in mind (§ 91a); also various *Aberrant forms of Pneumonia* (§ 91b).

TABLE IX.—*Differentiation between*

	LOBAR OR CROUPOUS PNEUMONIA.	LOBULAR OR BRONCHO- PNEUMONIA.
<i>Onset</i> . . .	Sudden, with rigors . . .	Gradual, and preceded by bronchitis.
<i>Course of</i> <i>Temperature</i> .	Continuous . . . .	Remittent.
<i>Defervescence</i> .	By crisis 7th day . . .	By lysis in 3 to 4 weeks.
<i>Percussion</i> .	Dulness in one lung, usually the base.	Scattered patches of dul- ness in both lungs.
<i>Auscultation</i> .	(i.) Fine crepitations (ii.) Consolidation-signs in a day or two.	(i.) Fine crepitations, and consolidation-signs, over dull areas; though ob- scured by rhonchi and bronchitic râles.
<i>Sputum</i> . . .	Rusty . . . . .	Frothy and muco-purulent.
<i>Respiration</i> .	Pulse-respiration ratio 2 : 1.	No marked difference of pulse-respiration ratio.

*Prognosis.* The case mortality varies from 20 to 40 per cent. in hospital cases. The usual mode of termination is by heart failure. Much depends on the position and extent of the lesion, which is graver when both lungs are involved, or when the disease attacks the apex. The reason of this is that apical pneumonia usually occurs in a lung already damaged by tubercle. A lethal termination may be anticipated with marked cyanosis, a typhoid condition, scattered râles over both bases (indicating œdema), with lowered temperature. The absence of the usual increase in the leucocytes is also said to be an unfavourable sign. It is also graver at the extremes of life, in alcoholics, and in debilitated persons; but robust men in the prime of life often succumb, although the prognosis is generally stated to be good in healthy adults. As regards complications, meningitis is generally fatal, and endocarditis extremely grave. But of all conditions influencing the prognosis of Lobar Pneumonia a history of chronic alcoholism is, in my belief, the worst.

The *treatment* of pneumonia is mainly expectant; but the

chief indications are (1) to allay the inflammation, and (2) to watch the heart, and act accordingly. The disease cannot be cut short, but in the early stages, when the pulse is bounding and the fever of a sthenic nature, the inflammation is allayed by a mixture of ammon. carb. gr. v, liquor ammon. acetat. 3j., and tr. aconiti  $\mathfrak{m}$  v every four hours. In the later stages the heart must be carefully watched, and strychnine, digitalis, or large quantities of alcohol employed, according to the necessities of the case as indicated by the pulse and heart sounds. Sleep must be procured, and for this purpose a large dose of sulphonal may be given in hot soup, followed in an hour by a dose of potass. bromide. In the aged, hypodermics of strychnine (gr.  $\frac{1}{60}$ ) and atropin (gr.  $\frac{1}{120}$ ) are useful stimulants. Inhalations of oxygen are good in states of exhaustion. Locally, some (*e.g.*, Dr. Lees) employ ice-bags to the chest, but these require careful watching, on account of the depression that may be caused. Poultices properly applied are safer and more comforting. An anti-pneumococcic serum is on trial (see Serumtherapeutics, Chapter XVIII.). F. 53, or 54, may be useful.

§ 91a. **A Pneumonic form of Acute Pulmonary Tuberculosis**, or pneumonic phthisis, is sometimes met with. The symptoms resemble those of pneumonia, and like it may start suddenly with a rapid rise of temperature, and pain in the side; and the temperature may continue high for a week or so. The physical signs also resemble those of pneumonia. It differs from this disease, however, in the presence of tubercle bacilli in the sputum, and the temperature, instead of falling abruptly by crisis about the 7th day, gradually becomes intermittent, and the *course of the disease* becomes indefinitely prolonged for weeks. This is followed by physical signs of breaking down, purulent expectoration, night sweats, and *generally, death* in five to twelve weeks from exhaustion, hæmoptysis, or complications, such as pneumothorax (see § 98).

§ 91b. **Aberrant Acute Pneumonias** (Deuteropathic Pneumonia). We have seen that in pleurisy, acute pneumonia, and in other local inflammatory diseases of the lungs, the course of the malady is fairly definite, and the physical signs in the lungs are characteristic and methodical. But it is important to remember, on the other hand, that these same conditions may occur secondary to, and as part of, some general disorder. In these the physical signs are frequently irregular; and it may not be possible to arrive at a diagnosis, except by passing in review the whole history of the case, and by making a thorough and systematic examination of all the other organs. Instances of this latter, or eccentric group of pneumonias, are met with secondary to enteric fever, cancer of the lung, acute glanders, anthrax, syphilis of the lung, and actinomycosis.

The practical outcome of these considerations is that when a case of pneumonia, or other apparently local inflammatory condition, is *irregular* in its physical signs, or its clinical history, we probably have to do with



a condition which is secondary to one of the conditions just mentioned, or to some general disease, such as enteric fever, scarlatina, pyæmia, or other general infective disorder.

III. *The illness has come on somewhat GRADUALLY, there is cough with frothy expectoration. The physical signs of CONSOLIDATION are SCATTERED and accompanied by signs of bronchitis—the disease is probably BRONCHO-PNEUMONIA.*

§ 92. **Acute Lobular Pneumonia**, or Broncho-pneumonia<sup>1</sup> is also an acute parenchymatous inflammation of the lungs, but it runs a very different course to that of acute lobar pneumonia. The inflammatory process occurs in small patches, scattered unequally throughout both lungs; and it is accompanied by bronchitis, hence its name. The disease in adults is tuberculous; only in children is it non-tuberculous.

The *constitutional symptoms* come on more gradually in this disease. The temperature is remittent, about 100° in the mornings and 101° to 103° in the evenings, accompanied by cough, dyspnœa, and frothy spit. The pulse is rapid, but the pulse-respiration ratio is not altered to anything like the extent of that in lobar pneumonia; and the face is generally pale instead of flushed. The fever is maintained by the fresh implication of neighbouring lobules for about three to six weeks or longer.

*Physical signs.* When the patches of consolidation are small, there may be no dulness on percussion, but only tubular breathing. But when they are of moderate size signs of consolidation (p. 169) can be made out. The chief auscultatory signs in children consist of *intensely loud*, “consonating,” râles and rhonchi.

*Etiology.* Broncho-pneumonia occurs at all ages; but is *especially frequent in young children*. It often accompanies measles and whooping cough, typhus and typhoid. The form occurring in adults is almost always of tuberculous origin.

*Diagnosis.* *Chronic phthisis* is limited to the apex at first and runs a characteristically chronic course. The pulmonary signs of *measles*, *whooping-cough*, and *bronchitis* resemble broncho-pneumonia, in its early stages, and it may not be easy to diagnose these several diseases until the rash of the one or the whoop of the

<sup>1</sup> Also called Catarrhal Pna. from the nature of the inflammatory process. The term Lobular Pna. is derived from its scattered distribution among the lobules of the lung tissue, some of which are attacked, and some exempt, giving a mottled greyish appearance on section.

other appears. The constitutional symptoms in acute bronchitis are much less severe. The diagnosis from *acute miliary tuberculosis* may be very difficult, but the tubercle bacillus occurs in the sputum of tuberculosis. The diagnosis from *lobar pneumonia* is given in tabular form above (p. 187).

*Prognosis.* The case mortality in children under five varies from 30 to 50 per cent. (Osler), being more fatal according to the tender years of the child. The strength of the patient and the duration of the disease are leading factors in the prognosis. If the patient is debilitated, especially if the environment is unfavourable, he soon becomes a prey to the tubercle bacillus, and the case rapidly runs on to phthisis (*q. v.*). Similarly, the longer the case lasts, the more likely is it to have a fatal termination, and, in adults, this is very often the case. Broncho-pneumonia is nearly always secondary, and the third leading factor in the prognosis is the nature of the antecedent disease. When a child, weakened by a *prolonged* fever, is attacked, the prognosis is very grave, but after whooping cough and measles it is much more favourable. Nevertheless children often recover in apparently hopeless cases.

*Treatment* resembles that of lobar pneumonia, but stimulants are indicated from the outset of the disease; two drops of brandy for every month of an infant's age may be given every second hour. Children should be placed in a steam-tent, and small frequent doses of tr. belladonna administered. For adults, the pain and incessant cough may require opium, best given as Dover's powder; and poultices applied to the back give considerable temporary relief. The chest afterwards may be covered with a cotton-wool jacket. If the symptoms become more distressing and the cough and dyspnoea increase, stimulating expectorants should be ordered; and, if the cough continue difficult, an emetic may even be given. For the reduction of the hyperpyrexia, cold sponging may be adopted, especially if cerebral symptoms are present.

We now turn to the **acute disease with hyper-resonance on percussion**—viz., Pneumothorax. We must bear in mind that an acute disease may supervene upon a chronic condition accompanied by hyper-resonance, *e.g.*, when acute bronchitis supervenes on emphysema. See Table, p. 220.

*The patient is in MARKED DISTRESS which has come on SUDDENLY. On examining the chest the BELL-SOUND is elicited—the disease is PNEUMOTHORAX.*

§ 93. **Pneumothorax** is a term applied to air in the pleural cavity, the air having gained admission by perforation of the pleura, either from within or from without. The air is almost invariably accompanied by pus, and the condition is then known as pyo-pneumothorax ; if accompanied by serous effusion, as hydro-pneumothorax (Fig. 48).

The *symptoms* of the onset of the condition differ according to the condition of the lung, *i.e.*, whether it is fairly healthy or is widely diseased. (a) When pneumothorax occurs in the less affected of the two lungs—the other side being extensively diseased—the symptoms are very urgent, and consist of severe pain in the side, attended by great dyspnoea, shallow, quick breathing, cyanosis, and some degree of collapse, with sweating, lividity, and a weak pulse. (b) In other cases, where pneumothorax comes on in a lung which is already much diseased, the onset may be hardly noticed.

The *physical signs* consist of : (i.) A

bulging on the affected side ; (ii.) diminished vocal fremitus ; (iii.) hyper-resonance on percussion (unless there is very great distension, when the note may be dull) ; (iv.) on auscultation the respiratory murmur may either be inaudible or amphoric ; the vocal resonance is usually diminished, but pectoriloquy and bronchophony are sometimes present. The “bell” sound may be elicited on tapping the chest with two coins in one position, and listening with a stethoscope in another. When fluid is also present, and this is usual, metallic tinkling is heard. The *Succussion-splash*, when it can be elicited without damage to the patient, is the most characteristic sign of hydro-pneumothorax, a fact which was well known to

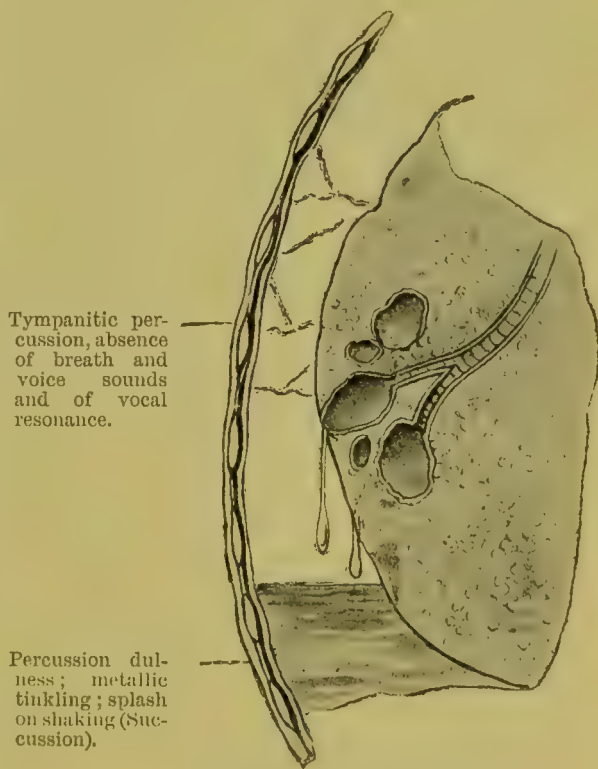


Fig. 48.—Diagram of HYDROPNEUMOTHORAX.

Hippocrates.<sup>1</sup> It may be obtained by placing one's ear against the chest, and shaking the patient's body to and fro.

*Etiology.* (i.) The commonest cause undoubtedly is advanced phthisis, when a cavity bursts into the pleura. (ii.) The converse process may take place in empyema, when the pus bursts into the lung. (iii.) A fractured rib may lead to perforation of the pleura. (iv.) Less common causes are gangrene of the lung, abscess connected with the spine, or an ulcer of the stomach or œsophagus, bursting into the pleural cavity. (v.) It rarely happens in healthy people, although cases have been recorded.<sup>2</sup>

*Prognosis.* The occurrence of pneumothorax is always very grave. It is difficult to estimate its case mortality, because death may be sometimes produced by the condition of the lung apart from the accident, but about half die within the first week, and some in a few hours, from shock or suffocation, when the lung on which the patient has been mainly dependent gives way. Only about 10 per cent. of all cases taken together ultimately recover. The *immediate* risk depends upon the urgency of the dyspnœa and cyanosis, the state of the other lung, the patient's general health, and the cause of the accident. As regards the *cause*, the pneumothorax that results from late phthisis or gangrene of the lung is very fatal; but that which occasionally complicates whooping cough, pneumonia, early phthisis, and injury, often results in recovery. Certain it is that the longer the patient lives after the onset of the pneumothorax, the better is the prognosis for ultimate recovery. Death usually occurs from shock and suffocation, as mentioned above; or from asthenia, due to the prolonged discharge and lung mischief.

*Treatment.* A hypodermic of morphia is desirable for the pain, and stimulants for the collapse. The question of paracentesis is an important one. It has been done with advantage when great distension is present, as indicated by marked displacement of organs, extreme pain and discomfort. When fluid is present it should be drained and treated as in empyema, unless it is very freely expectorated. In other cases it is inadvisable to aspirate or operate; especially in phthisical cases, which should be left to the "*vis medicatrix naturæ*."

*There is one disease of the lungs which belongs neither to the acute nor to the chronic category, but is PAROXYSMAL, occurring in attacks of sudden onset, usually WITHOUT ELEVATION OF TEMPERATURE—ASTHMA.*

§ 94. **Asthma** is characterised by sudden paroxysmal attacks of very severe dyspnœa, the inspiratory effort being short, the expiratory prolonged and accompanied by much lividity and distress. Asthma is liable to complicate chronic bronchitis, but it is important here to draw attention to the frequent error which is made by regarding all exacerbations of chronic bronchitis as asthma.

*Symptoms and clinical history.* The leading characteristic of this disease is its paroxysmal nature. A person who is subject to asthma may be perfectly well at one minute, and half-an-hour later may be seized with the most violent dyspnœa. It often commences in the early morning, the patient awakening with a feeling of tightness of the chest; he gasps for breath, and clings to surrounding objects in order to fix the extraordinary muscles of respiration. Each attack lasts from a few hours

<sup>1</sup> "De Morbis," Lib. II., Cap. XVI.

<sup>2</sup> Transactions of the Medical Society, vol. xx., 1897, p. 120.



to a few days, and then, without apparent reason, the patient suddenly recovers his normal and healthy condition.

There are many curious and unexplained features in connection with this malady, one of the most interesting being the tendency to skin eruptions (especially urticaria and the various forms of erythema); and another, the fact that these eruptions may alternate with the attacks of dyspnoea. Various other neuroses, and even attacks of insanity, may alternate in the same way. The paroxysms of asthma are occasionally preceded or ushered in by violent attacks of sneezing, by itching, or by the passing of large quantities of limpid urine. Sometimes an attack is terminated in this way.<sup>1</sup>

*Physical signs.* On inspection the chest is seen to be maintained in a position of inspiration, undergoing but little expansion with the short inspiratory efforts. The percussion note may be unaltered, but, after many attacks, emphysema supervenes, with consequent hyper-resonance. On auscultation the short inspiratory effort is feeble and scarcely audible; expiration prolonged. Loud rhonchi replace the normal vesicular murmur; and often coarse râles, owing to the accompanying bronchitis.

*Etiology.* Some regard the asthmatic attack as a series of spasmodic attempts on the part of the diaphragm, intercostal muscles, and extraordinary muscles of inspiration to overcome some obstruction to the entry of air. But the central fact, which alone explains all the symptoms, is a narrowing of the bronchial tubes. This is probably due to spasm of the involuntary bronchial muscles, which is attended by hyperæmia of the submucosa and swelling of the mucous membrane. Some hold that the latter is the primary condition, and that the disease is therefore an angioneurosis.

Among the *predisposing* causes we find :—(i.) A neurotic family history. Careful inquiry may reveal asthma or other neuroses, especially those so-called functional diseases of the nervous system connected with the involuntary muscular system, such as attacks of flushing and shivering, faints, and the like. (ii.) Asthma may occur at any *age*, but true asthma nearly always makes its first appearance soon after the age of puberty. (iii.) Any previous lung disease, especially chronic bronchitis, may predispose to asthma. Malaria, gout, and other constitutional conditions are often associated with it.

Among the *exciting* causes of an attack may be mentioned :—(i.) Certain atmospheric conditions which are ill-understood and often appear to be most contradictory. Thus, I know one patient who is free from asthma in London, but develops an attack immediately she seeks a high altitude. Another always develops an attack when she enters London. Some find the sea relieves them, others that a seaside place determines their attacks. (ii.) Reflex causes, such as derangement of the alimentary canal, and dietetic indiscretions (*e.g.*, the eating of cheese or fish), will often determine an attack; and so also will (iii.) dust and irritating particles.

*Diagnosis.* The diagnosis usually presents no difficulty. The paroxysmal occurrence of the disease is quite characteristic. *Chronic Bright's* disease is manifested sometimes in its very earliest stages by paroxysms of dyspnoea resembling asthma. These are especially apt to occur at night.

<sup>1</sup> These facts point possibly to an affection of the general vaso-motor system similar to that in the pulmonary system which produces the spasmodic dyspnoea.

*Cardiac Asthma* is a term which is loosely applied to the paroxysmal attacks of dyspnoea which accompany heart disease. It is differentiated from true asthma (i.) by its frequent recurrence independently of the causes of true asthma, and (ii.) by the evidences of the cardiac mischief.

*Prognosis.* The disease of itself does not shorten life, but tends to produce emphysema, bronchitis, and their attendant evils. Children may grow out of the disease; adults never lose it completely. The severity and frequency of the attacks are our only guides to prognosis.

*Treatment.* (a) *Before the attack.* Various remedies have been tried. Mentioned in the order in which I have found them most useful there are: tr. lobelia, ether, belladonna, hyoscyamus, opium in small doses, and pyridin, a remedy introduced and strongly advocated by Germain See. The diet during the attack should be the lightest possible; milk alone is best. Various inhalations are sometimes useful, either for the prevention or relief of an attack; e.g., the vapour from a teaspoonful of turpentine and chloroform in equal parts, or the fumes of paper prepared with a strong solution of nitrate of potash, or the inhalation of amyl nitrite. Various patent remedies are in great repute, especially Himrod's Asthma Powder. Various other preparations, in the form of cigarettes of stramonium, pot. nit., and belladonna, are used. Experimentally on animals, I have found pilocarpin relaxes the bronchial muscles, and it would be worth trying, therefore, in asthma.

(b) *Between the attacks.* The effect of locality on the disease can only be ascertained by experience, and, as above mentioned, it is impossible to foretell what effect a particular climate will have. As a rule, though with many exceptions, town air and fogs are detrimental. To prevent an attack, special attention should be directed to the diet. Light nourishing food should be advised; and the avoidance of solids after two o'clock in the day. Iodide of potassium administered for a long period of time certainly tends to ward off attacks in some patients. Arsenic also is very useful. Potass. bromide at bedtime may ward off an attack. The nose should be examined for polypi, etc., and these, as possible causes of irritation, must be removed.

#### CHRONIC DISEASES OF THE LUNGS AND PLEURÆ.

§ 95. *Classification.* Chronic disorders of the lungs and pleuræ may follow an acute attack of the conditions described in the previous sections, as when chronic bronchitis and emphysema succeed attacks of acute bronchitis. But many of the chronic diseases of the lungs, such as pulmonary tuberculosis, start insidiously, and attention may not be directed to the lungs for a considerable time.

The chronic diseases, like the acute, may be classified for clinical purposes, according to the results of percussion. It is convenient in actual practice, although unscientific, from the point of view of classification, to make a subsidiary group in which the sputum is of a highly offensive character.

a. **Chronic Disease** in which the **Percussion Note** is **unaltered**,

I. Chronic bronchitis . . . . . § 97

b. **Chronic Diseases** attended by **Dulness on Percussion**,

The *commoner* disorders presenting dulness in regular and *defined areas either at base or apex*,

I. Chronic phthisis . . . . . § 98

II. Hydrothorax . . . . . § 99

III. Pulmonary congestion (or œdema of the lungs) § 100

The *rarer* diseases, having *irregular and scattered* areas of dulness,

IV. Interstitial Pneumonia . . . . . § 101

V. Thickened Pleura . . . . . § 102

VI. Cancer and other neoplasms . . . . . § 103

VII. Collapse of the lung tissue . . . . . § 104

VIII. Syphilitic Disease of the Lung . . . . . § 105

(IX. Mediastinal Tumours) . . . . . § 57

c. **Chronic Diseases** attended by **Hyper-resonance**,

I. Emphysema . . . . . § 106

II. Pneumothorax<sup>1</sup> and various other conditions,  
in which the hyper-resonance is not the  
leading or constant feature . . . . . § 93

d. **Diseases** recognised by the **offensive character of the Sputa**,

I. Bronchiectasis . . . . . § 107

II. Gangrene of the lung . . . . . § 108

III. Abscess of the lung . . . . . § 109

§ 96. **Method of procedure.** The routine examination is conducted as in acute disorders (§ 84), viz., after ascertaining the leading symptom, and the history of the illness, we proceed to Inspection, Percussion, Auscultation, and Palpation. In percussion, remember to keep the hand flat and firmly pressed against the chest, while it is struck by one or more fingers of the other hand, used as a hammer, and with a staccato stroke. Remember also that the note is normally dull over the mammae in most women, over the scapulae in muscular men, and that it is slightly lower-pitched at the right than the left apex. The chest must of course be stripped.

<sup>1</sup> Pneumothorax sometimes comes on acutely, but it is more often part of a chronic disease.

GROUP a. The patient's symptoms point to **chronic disease of the lungs**; and on examining the chest there is **no alteration in the percussion note**.

I. *The patient has a chronic cough; there is no elevation of temperature, and on auscultation RHONCHI and RÂLES are heard over the chest. The disease is CHRONIC BRONCHITIS.*

§ 97. **Chronic Bronchitis** is a chronic inflammation of the bronchial tubes. It may be chronic from the beginning, or it may supervene on repeated attacks of the acute disorder.

*Symptoms.* A patient with chronic bronchitis and—its usual sequel—dilated r. heart presents a very typical appearance. Stout in build, with short thick neck, of florid complexion, short of breath, wheezy respiration, and pulsating jugular veins, he presents an aspect which can be recognised at once. The clinical history extends over many years, with alternate diminution and aggravation of the symptoms. The cough is usually present during the winter, and improves as the weather gets warmer. The constant coughing and straining to bring up the secretion results sooner or later in generalised emphysema. In later stages the cough continues all the year round, and finally an attack of capillary bronchitis, œdema of the lung, or some intercurrent malady, throws a little extra strain upon the over-burdened r. heart, and death ensues. There are, as a rule, no febrile or constitutional symptoms.

The *physical signs* vary with the amount of secretion present, the amount of the complicating emphysema (§ 106), and bronchiectasis (§ 107). In cases of long duration the chest is barrel-shaped (emphysematous, § 77). Rhonchial fremitus may be felt on palpation. On percussion there is never any dulness; and the note is hyper-resonant in proportion to the emphysema present. On auscultation sibilant and sonorous rhonchi and bubbling râles can be heard; and crepitations at the base, due to œdema, may be present.

There are four recognised varieties of this disease: (i.) Bronchitis with *winter cough*, attended by slight or abundant expectoration, mucous, or muco-purulent, sometimes fibrinous, sometimes containing streaks of blood. (ii.) *Dry Bronchitis* is attended by a frequent cough and soreness of the chest, but little or no



secretion ; it is of a very obstinate character, and occurs mostly in elderly people of a gouty diathesis. (iii.) *Bronchorrhœa* is recognised by the expectoration, which is of a thin, clear, or thick and ropy nature, very abundant and devoid of air. (iv.) *Fœtid Bronchitis* may occur in the later stages, and marks the onset of bronchiectasis. The sputum is very fœtid from time to time (see bronchiectasis, § 107).

The *diagnosis* of Ch. Bronchitis is not usually difficult. It may be readily diagnosed from *chronic phthisis* by the appearance of the patient, by the absence of hectic fever and emaciation, and by the absence of the tubercle bacillus from the sputum.

*Etiology.* Chronic bronchitis may occur at any age, but is more common in elderly people. Sometimes, as before stated, it follows repeated attacks of acute bronchitis, but it may be chronic from the beginning. It often affects plethoric subjects, especially those of a gouty habit ; and it is one of the recognised complications of Bright's disease. It is a frequent sequel to cardiac valvular disease, more especially disease of the mitral orifice. It may complicate other diseases of the lungs, especially phthisis ; and may be a sequel of the acute specific fevers, especially measles and enteric fever.

*Prognosis.* Patients with chronic bronchitis seldom entirely recover, though they may live for a great many years ; and if the heart is fairly healthy and care be taken to avoid exposure, life is not very materially shortened. The co-existence of gout, Bright's disease, and cardio-vascular degeneration make the prognosis somewhat less favourable. The condition of the lungs is not so much a guide to prognosis as the condition of the heart. This indeed is the point around which the prognosis centres ; and the untoward symptoms which render the prognosis grave are thus referable to the heart, viz., considerable dilatation of the r. heart with evidences of cardiac failure, such as dropsy, rapid irregular pulse, great breathlessness, cyanosis (see § 53).

*Treatment.* The extreme frequency of the disorder renders the treatment a matter of considerable importance. In severe cases the patient must be confined to one room at a uniform temperature of 62° F. day and night. When the mucous membrane is dry and irritable a steam kettle gives great relief ; it must be

kept constantly going, not used intermittently. In slight cases, however, the patient can go about, but chill and exposure should be avoided. The important question of when a patient may go out must depend largely on the weather—cold and moisture, especially when in combination, are especially injurious.

The indications as to treatment are:—(i.) to stimulate the relaxed mucous membrane with such remedies as am. carb., senega, squills, etc. (ii.) When the cough is dry, soothing remedies, such as bromides, codeia, and tr. camph. co. should be given, or remedies directed to promote the secretion, such as ipecac., ammon. chlor., potass. iod., and alkalies (the last two



Fig. 48a.—Bronchial Cast.

especially in rheumatic or gouty cases), may be employed. (iii.) When the sputum is very abundant we should endeavour to diminish secretion by such remedies as the balsams (tolu and peru), tar preparations (creosote, guaiacol, petroleum), turpentine, camphor, senega, etc., given either internally or in the form of inhalations. For the latter, tar, creosote, and terebene may be used. Counter-irritants to the chest, *e.g.* turpentine, camphor, or eucalyptus, are very popular with some. (iv.) When there is much

spasm of the tubes, lobelia, iodide, and other asthmatic remedies are to be tried. (v.) Cardiac tonics and stimulants are called for sooner or later where dyspnoea and other cardiac symptoms are present. (vi.) In cases with a gouty taint Ems water each morning, and small doses of iodide may be added to the other treatment, and the emunctories may be aided by sipping hot water morning and evening and at intervals during the day.

§ 97a. **Plastic Bronchitis** is inflammation of the bronchi with the formation of fibro-plastic casts which are expectorated.

*Symptoms.* The symptoms consist of (i.) Violent attacks of coughing, with expiratory dyspnoea, followed by (ii.) The expectoration of a fibrinous cast of a bronchus (*vide* Fig. 48a). (iii.) The patient generally suffers from chronic bronchitis, and a little hæmoptysis may follow the expulsion of a cast. (iv.) Sometimes there are no constitutional symptoms, but slight pyrexia, and in some cases even rigors, may be present. Such

symptoms supervening in a case of chronic bronchitis lead us to suspect the condition.

*Physical signs* may be absent. If present, they are those of an obstructed bronchus—an absent or diminished respiratory murmur, accompanied possibly by impaired percussion note. Whistling rhonchi or “flapping” sounds may be heard.

*Causes.* The disease is twice as common in men as in women. It may occur at any age in subjects of chronic bronchitis.

*Prognosis.* The condition is more serious than bronchitis. Two varieties have been described:—(1) An acute form lasting for some weeks, and (2) a chronic form occurring at intervals, for years, in the course of chronic bronchitis. Each attack may last for some weeks, and the casts be coughed up daily. The condition occasionally leads to a fatal issue from dyspnoea, as when a large cast cannot be brought up.

The *treatment* differs but little from that of bronchitis. The removal of the membrane may be promoted by the inhalation of limewater, atomised by means of a spray, which is used with a view to dissolve the mucin in the cast. Various oils (*e.g.*, creosote oil 1 in 40) have been injected as solvents, but the results have not been very promising.

GROUP b. We now turn to those Chronic Diseases of the Lungs which are accompanied by **dulness on percussion**.

(a) The more *common* diseases, in which the dulness occurs in regular and fairly DEFINED AREAS at base or apex, are : I. CHRONIC PULMONARY TUBERCULOSIS, II. HYDROTHORAX, and III. PULMONARY CONGESTION OR ŒDEMA.

I. *The patient complains of gradual emaciation and perhaps cough ; on examination of the chest SIGNS OF CONSOLIDATION may be found, most marked at the APEX of the lung. There is INTERMITTENT PYREXIA, and the sputum may contain the tubercle bacillus—the disease is CHRONIC PULMONARY TUBERCULOSIS (Phthisis).*

§ 98. **Chronic Pulmonary Tuberculosis** (Phthisis) may be defined as a wasting disorder due to tuberculosis of the lungs. The word phthisis is objectionable because it only indicates one of the symptoms, *viz.*, the wasting ( $\phi\theta\acute{\iota}\omega$ —to waste). In view of the fact that this disease is the chief cause of death in Great Britain, the importance of the subject cannot be over-estimated. The average annual number of deaths in London alone, for the decade 1889-98, was 8,453. The disease was formerly regarded as due to a kind of neoplasm, consisting of little round, nodular growths, “tubercles,” scattered throughout the lungs, which are made up of a large number of small round cells, epitheloid cells, and giant cells. Owing to the discoveries of Koch we now know that these

little nodules are only the inflammatory manifestations which arise consequent on the irritation of a minute bacillus (the tubercle bacillus), and that the disease is primarily due to the ravages in the economy of this bacillus and its toxic products, and secondarily, to the supervention of other infective processes, and especially those due to pyogenic organisms invading the lungs.

It is customary to describe the anatomy in three stages. It is now generally believed that tuberculosis of the lungs begins as a tuberculous endo-bronchitis, due to the settling of the microbe in



III. Cavitation.

II. Consolidation.

I. Congestion.

Fig. 49.—THE THREE STAGES OF PULMONARY TUBERCULOSIS—SOMETIMES PRESENT TOGETHER IN THE SAME LUNG.

one of the smaller bronchial ramifications. It has been shown by Birch-Hirschfeld, who took metallic castings of the bronchial tubes, that the reason the microbe settles at the apex is because in this situation there is, as it were, a “dead end,” in which air is not so readily changed as in other situations. As a consequence, any dusty particles containing the bacillus which are inhaled

and reach this situation, settle down, and there set up an irritation, resulting in a small localised ulceration of the mucous membrane. The *congestion* which takes place around these constitutes (a) The first stage. (b) In the second stage there is considerable cell proliferation filling up the air cells and resulting in the formation of nodules consisting of granulomatous material in the neighbourhood of the primary mischief. This is the stage of *consolidation*. (c) The third stage is one of *breaking down*. Owing to the indolent character and low vitality of the new cell formation, it caseates and softens, becomes the seat of pyogenic organisms, destroys the air cells and forms smaller or larger cavities. Thus we have three stages: (a) congestion, (b) consolidation, and (c) breaking down, with the formation of cavities (Fig. 49).



*Symptoms.* The disease is essentially a chronic one, and its onset is very insidious. It is always more amenable to treatment in the early stages, and since the introduction of modern methods of treatment an early recognition of the disease has come to be of paramount importance.

(a) *Prodromal Stage.* Phthisis has six modes of onset, which in order of frequency are as follows:—(i.) progressive weakness, attended perhaps by cough; (ii.) hæmoptysis<sup>1</sup>; (iii.) dyspepsia; (iv.) laryngeal tuberculosis; (v.) dry pleurisy; (vi.) acute pneumonia (§ 91a), bronchitis, or broncho-pneumonia. Among the earlier *general symptoms* which should make us suspect the invasion of tubercle are unexplained debility, attended by languor and anæmia on the one hand; or loss of weight, with unexplained dyspepsia, or slight elevations of temperature in the evening on the other. The temperature is an indication of the very greatest importance, for *no ACTIVE tubercular process can take place in any part of the body without the occurrence of some pyrexia*, however slight. The type of this pyrexia is equally distinctive, for it is of an *intermittent character*, being normal in the morning and raised in the afternoon or at night; in rare instances *vice versâ*. If we have any suspicion of tubercle, the temperature should be taken every two hours, so that we may not miss any slight access of temperature during the day. Allbutt has pointed out that a premenstrual elevation of temperature sometimes occurs. In the early stage the patient may not be aware of the feverishness, though generally he feels a chilliness in the evening, and as the disease progresses night sweats form one of its most characteristic features.

The later symptoms of the disease are largely due to the action of other organisms than the tubercle bacillus. The clinical manifestations of these “mixed infections” (which consist chiefly of pyogenic and influenza microbes) are not always obvious, but many hold that whenever the temperature rises above 100·5° or 101° F. it is due to a superadded infection of this kind. It adds considerably to the gravity of a case, and its prevention, by fresh air, cleanliness, and a hygienic mode of life is important.

The *physical signs* accompanying the prodromal stage are

<sup>1</sup> Early hæmoptysis of a very profuse kind may occur before any physical signs are discoverable.

necessarily somewhat vague and difficult to detect. The patient's chest should be thoroughly stripped and he should be taken to a room where perfect quiet prevails ; and if with the above symptoms we find weak or harsh breathing at one apex—especially if this is accompanied by an occasional single sibilant râle—we may be fairly certain that the disease is developing. Persistent inspiratory “sticky” clicks at one apex are very suggestive, if not pathognomic of early phthisis. It is important to auscultate while the patient coughs, for râles not previously audible may thus become evident. The signs just named can often be heard best at the apex, behind, by placing the patient's hand on his opposite shoulder and listening to that part of the lung, just external to the bronchi, which will thus be *uncovered by the scapula*. Fine crepitations may be heard in that situation weeks before any signs can be discovered at the apex in front. Sometimes, later on, we are led to detect phthisis by an undue loudness of the *heart* sounds at the apex of one lung. Absence of dulness, like the absence of bacilli, is not evidence of the absence of tubercle.

The *sputum* should be repeatedly examined for tubercle bacilli. Although the disease can be diagnosed in their absence, by means of the above physical signs, it is always useful to employ this confirmatory test. The absence of bacilli, even after a series of examinations, does not indicate the absence of phthisis ; but their presence establishes the disease beyond dispute. Another confirmatory test is the use of Koch's “old” tuberculin (see p. 209 ; and Chapter XVIII.), which, although not much approved of in England, is extensively used in Germany. It is followed by a definite reaction in phthisical subjects, even in the early stage.

(b) The *stage of consolidation* and (c) the *stage of softening* and cavitation may be dealt with together. The symptoms, physical signs, and the corresponding lung changes are given in the form of a table for the purposes of convenience. The physical signs usually begin at the apex, and are generally to be best heard at the back, as above mentioned ; sometimes at the apex of the lower lobe. From this position they extend downwards, and thus it is possible in the same patient to recognise in advanced cases the signs of the third stage, or cavitation, at the apex ; below these, signs of consolidation ; and below these, signs of congestion (as in

Fig. 49). Such a condition indicates considerable activity. Many accessory signs may be mentioned: enlarged heart area due to retracted lung, hæmic heart murmurs due to anæmia, clubbed fingers in chronic cases of long duration, etc. Extensive tuberculous disease may sometimes exist with but little constitutional disturbance, and on the other hand, considerable disturbance of health may be present, without any abnormal physical signs—depending, partly, on the distance of the lesion from the surface of the lung.

TABLE X.—THREE STAGES OF PHTHISIS.

<i>Anatomy.</i> (See Fig. 49, p. 200.)	<i>Physical Signs.</i>	<i>Symptoms.</i>
<i>a.</i> CONGESTION of lung tissue, consequent on invasion by tubercle bacilli.	At apex of the lung, (i.) Feeble R.M., with occasional fine crepitation heard at end of inspiration; or (ii.) Unduly harsh breathing, with a prolonged expiration.	(i.) Increasing languor on exertion; (ii.) Slight morning cough; (iii.) Slight rise of temperature; (iv.) In some cases hæmoptysis.
<i>b.</i> CONSOLIDATION — due to the hyperplasia, cell infiltration, and the fusing together of the tubercular foci.	Over diseased part, usually at apex, are: (i.) Impaired movement; (ii.) Flattening; (iii.) Increased vocal fremitus; (iv.) Dull percussion note; (v.) Bronchial or tubular breathing; (vi.) Bronchophony (increased V.R.).	(i.) Weakness and emaciation increase; (ii.) Temperature markedly higher in the evening; (iii.) Night sweats; (iv.) Anæmia.
BREAKING DOWN and EXCAVATION.	Signs as in <i>b</i> , <i>plus</i> , 1. Moist clicking râles, and later on— 2. Signs of presence of cavity: (i.) Cavernous breathing. (ii.) Post-tussic suction. (iii.) Pectoriloquy. (iv.) Râles with metallic tinkle.	All the above symptoms aggravated: (i.) Cough distressing, with quantities of nummular expectoration; hæmoptysis may be profuse; (ii.) Temperature high, and with wide range; (iii.) Sometimes diarrhœa, etc.

The presence or absence of A CAVITY is in the majority of cases impossible to diagnose with certainty. The percussion note is usually dull, but varies with circumstances. Thus the note is resonant when (i.) the cavity is very large, or lies very superficially; and (ii.) there is not a great amount of consolidated lung tissue between the cavity and the chest-wall. When the cavity is large and superficial, and the communicating bronchus remains patent, a characteristic note, almost tympanitic, is obtained on percussion whilst the patient keeps his mouth open. This is known as the "cracked-pot" sound (*bruit de pot fêlé*). Many attribute most importance to the sign known as post-tussic suction. To elicit this sign, the stethoscope is applied over the suspected cavity, the patient is told to cough, and immediately after the cough a characteristic swishing sound is heard, due to the sucking of air into the cavity. This may be accompanied by copious râles.

The *diagnosis* of the disease is not difficult excepting in the early stages, and in the absence of bacilli from the sputum. (i.) Various other causes of hæmoptysis may have to be differentiated (see § 76); (ii.) various other causes of anæmia may have to be eliminated (Chapter XIX.); (iii.) when the condition begins with dyspepsia it is very liable to be overlooked unless the physician is aware of this mode of commencement; (iv.) other causes of cough may be mistaken for phthisis (see § 73); (v.) various laryngeal affections may have to be eliminated (§ 122); (vi.) simple pleurisy is only rarely mistaken for tuberculous pleurisy because the latter is of such slow insidious onset, and its tendency is not so much to the effusion of fluid as to the formation of adhesions.<sup>1</sup> (vii.) When it supervenes on bronchitis or broncho-pneumonia our only clue to the fact consists in a delayed convalescence, together with the persistence of râles; and the fine clicking râles of phthisis are quite distinctive to the experienced ear. In the later stages of the disease the differentiation from the other causes of percussion dulness is not difficult (Table, p. 195).

*Etiology.* In phthisis, as in other microbial disorders, there are, on the one hand, predisposing causes which relate to the patient (*i.e.*, the soil on which the bacillus grows) and his powers of resistance; and, on the other hand, exciting causes which relate to the microbe itself. But phthisis differs most markedly from all other microbial disorders in this, that the predisposing factors play the leading part. If the "soil" is not suitable, *i.e.*, if the person is

<sup>1</sup> According to Fiedler 82 per cent. of apparently simple pleurisies with effusion, even of sudden onset, are in reality tubercular. This is confirmed by Burrs, Bowditch, F. R. Walters, and others. Indeed all pleurisies in strumous persons should be looked at with suspicion.



not predisposed by heredity or other cause, the bacillus will rarely grow. For these reasons its infectivity has been overlooked all these years. (1) Heredity is a potent cause, the individual being born with a predisposition to the disease. This factor, however, does not, as we shall see, occupy the prominent position which it was formerly believed to occupy. In a large proportion of cases no evidence of heredity is obtainable. (2) Both sexes are pretty equally affected, but the favourite age at which the disease usually supervenes is between 20 and 30. The patient may be attacked at any time of life, although it is very rare under two years. (3) Any condition of malnutrition may produce a predisposition to the bacillus invasion, whether it arise from deficient food, from hyper-lactation, from exhausting diseases such as diabetes, or the acute specific fevers, after which an attack of phthisis is by no means infrequent. It is a curious circumstance that pregnant women are not prone to the disorder, and a phthisical subject becoming pregnant will often improve until after her confinement, when an exacerbation of the disease will occur, which has usually a fatal result. (4) Unhealthy surroundings play a most important part in the production of phthisis, and indoor occupations such as those of lace-makers and city clerks are specially liable. (i.) A damp soil undoubtedly favours the production of the disease—even the dampness from faulty construction of a dwelling will do so. (ii.) A moist, hot atmosphere, such as exists in certain factories, favours the spread of the disease. (iii.) A dust-laden atmosphere, such as that of stonemasons, knife-grinders, tin and copper miners, fustian-cutters, is a potent cause of phthisis.

It is, however, an undoubted fact that mankind is naturally resistant to the tubercle bacillus. Birch-Hirschfield undertook a laborious investigation of 4,000 post-mortems, and he found that in 40 per cent. of these persons, dying from all manner of diseases, the lungs showed evidences of tubercle which had become quiescent and undergone spontaneous recovery. In view of these facts, and that most of the predisposing causes above mentioned are preventable, there is no reason why phthisis should not one day become as rare in England as leprosy is to-day.

*Prognosis.* (1) *Usual course and duration.* Phthisis is essentially a

chronic but progressive disorder, and until recently nearly all cases applying for treatment terminated fatally. The death-rate from phthisis in 1838 was 38 per 1,000, in 1892, 14 per 1,000 living. (Pollock, Discussion, Roy. Med. Chir. Soc., Dec. 1899.) Rapid cases may terminate in death in the course of three to six months. When the disease is indolent, and the patient resistant to the microbe, it may drag on for years. There are four modes of death, which in order of frequency are (1) asthenia, (2) hæmoptysis, (3) asphyxia from pneumothorax, (4) the occurrence of other complications.

2. The prognosis in reference to *causation* depends on :— (i.) Heredity. Unquestionably it takes a more favourable course and the process tends to be less active in cases where there is no family history of tuberculosis. (ii.) The age of the patient influences the course considerably, for it is much more rapid in the young than in people over 30. (iii.) The hygienic surroundings of a patient, as we shall see under treatment, make considerable difference to the course of the disease. Where the patient is well-to-do and can be removed from those conditions which have promoted the disease, he has a good chance nowadays of recovery ; but among the poor, who are forced to continue among their squalid surroundings and at their work, a fatal issue almost necessarily results. (iv.) Previous alcoholic excess diminishes the chance of recovery.

3. *Untoward symptoms.* (i.) Undoubtedly the most important feature is the temperature. Not only is an active tuberculosis evidenced by pyrexia, but the degree of fever, and still more the extent of the variations, are a fairly precise measure of the activity of the tuberculous process. (ii.) The condition of the lung is of course important. The presence of râles, as denoting softening and advancing disease, is unfavourable ; and their disappearance favourable, but the extent of lung involved is as important a factor in prognosis. Thus, a man in the third stage, with an apex cavity in one lung, and little disease elsewhere, has a better chance of recovery than one with slight tuberculous foci scattered through the lung. If both lungs show disease in the third stage recovery is rare, though health has been restored in some cases after prolonged treatment. (iii.) The general symptoms also aid us in recognising the rate of progress. When the weight is increasing, the temperature declining, and food is taken well, the

chances of recovery are good. (iv.) Early hæmoptysis does not affect the prognosis in any way, but occurring later in any quantity is apt to weaken the patient considerably.

4. *Complications.* The presence of complications is undoubtedly bad. The commonest complications are :—(1) Pleurisy, which is very frequent, but is often of a conservative nature, for adhesions may sometimes prevent pneumothorax ; (2) tubercle may occur in other parts—the peritoneum, meninges, and especially in the intestine, giving rise to ulceration and an exhausting diarrhœa<sup>1</sup> ; (3) the larynx may be affected either previously or subsequently, and undoubtedly it adversely influences the prognosis ; (4) lardaceous disease of the liver, spleen, and other organs frequently ensues, and this again constitutes an unfavourable element, as it cannot be removed ; (5) pneumothorax and pyo-pneumothorax may ensue from the bursting of a cavity into the pleura—fatal asphyxia *may* result (§ 93) ; (6) thrombosis of various veins is a less common complication ; (7) peripheral neuritis is now a recognised occurrence, sometimes very early in the disease ; (8) vomiting (see p. 208).

It is a good rule never to commit yourself to an opinion on any case of phthisis without first noting the effects of treatment.

*Treatment of Phthisis.* The subject of treatment will be dealt with under four headings—(a) remedial ; (b) symptomatic treatment ; (c) treatment by tuberculin ; and (d) the open air treatment. The indications of all treatment are to reduce the inflammation, to destroy the virus, to build up the strength, and to palliate the symptoms.

(a) The *remedial treatment* formerly in vogue was mainly directed to building up the strength by means of cod-liver oil, maltine, hypophosphites, and other tonics. These are still useful excepting when there is considerable fever present, or there are evidences of rapid breaking down. Guaiacol 20 grains (gradually increased), creosote, thymol, eucalyptus, and other antiseptics may be given at any time ; and a few years ago these were also administered by some as an injection into the lung. Perhaps the best of this kind of treatment is the use of a spray four times a day, lasting fifteen minutes, of formalin.<sup>2</sup>

<sup>1</sup> Diarrhœa may also occur as part of the hectic fever without any ulceration of the bowels.

<sup>2</sup> Dr. Larchner Green recommends the following formula :—(Formalin = 40 per cent. of Formic Aldehyde). Formalin 5j ; Glycerin 5iv ; Aq. Dist. 5v. Use as spray four times a day—fifteen minutes at each inhalation.—*Lancet*, Aug. 19, 1899, p. 521.

Counter-irritation was largely used to reduce the inflammation, the favourite being iodine or croton oil applied over the apex of the lung. Inhalation of antiseptics may be administered by Coghill's respirator. These measures were supplemented in wealthier patients by sea voyages, high, dry mountain air, and residence abroad during the winter, combined with a liberal dietary and general hygienic mode of life.<sup>1</sup> These various methods are still useful, but at the present time we have two powerful means of combating this lethal disease: viz., Koch's tuberculin, and the open-air treatment, see (c) and (d) below.

(b) *Symptomatic Treatment.* It will be seen that in the third stage there is not much hope of recovery, but even in the worst cases we can ameliorate the symptoms, and so ease the passage to the grave. (1) For the cough, tinct. camph. co. and other expectorants are not much use. The best cough mixture is one containing liquor morph., or better still codeia, in small doses with acid sulph. dil. Warm alkaline drinks promote expectoration. (2) Night sweats, which are often very profuse and exhausting, may be combated by atropine, zinc oxide, picrotoxin, and strychnine, especially the first named. Night sweats are said to be seldom troublesome if there be free exposure to fresh air. (3) The diarrhœa is also very exhausting, and must be combated with catechu, opium, intestinal disinfectants, and mineral acids. (4) Pleuritic pains may be eased by stupes, or painting with tinct. iod. (5) The concurrent dyspepsia must be combated in the usual way, but the vomiting is often a very troublesome symptom, and there are three kinds of vomiting which admit of three different methods of treatment. (a) If preceded by nausea, it points to disorder of the stomach, and should be treated by bismuth, etc., on the usual lines. (b) If the vomiting be preceded and caused by coughing it is a good plan to give hot drinks just before a meal in order to encourage the expectoration and get the paroxysms of coughing over before the meal is commenced. (c) If neither of these causes can be traced, the vomiting is

<sup>1</sup> Simple respiratory exercises, such as the following, designed to expand the chest form a useful adjunct: (i.) With the back against the wall fully extend the arms to the level of the shoulders slowly for eight times. (ii.) Continue the same movements until the arms meet above the head. (iii.) Start with the hands above and in front of the head, and bring them slowly down until the backs of the hands meet behind the body, at the level of the buttocks, the arms being rigid all the while. These should be done twice daily, gradually increased to five to six times daily.



probably due to irritation of the vagus, and may sometimes be relieved by opium. Sometimes vomiting is controlled by the will. (6) The treatment of hæmoptysis, pneumothorax, and laryngeal ulceration are dealt with elsewhere. (7) Now that the microbic origin of tubercle is established, the question of how far it is an infective disorder is keenly debated, but whether all cases take origin directly, or indirectly, from previous cases or not, true it is that if there were no bacilli there would be no consumption. Our preventive measures must therefore be directed (1) to the destruction of the microbe, and (2) to render those liable to the disease by heredity, resistant to the disease by a perfect state of health. The measures for the destruction of the bacillus are given in text-books on hygiene, but, briefly, the means consist of:—(1) The destruction of the sputum by collecting it into a disinfecting medium; and (2) the thorough disinfection of rooms occupied by phthisical persons. The larger preventive measures of stamping out the disease from a community are of the highest importance. How much improvement hygiene can effect in this respect is shown by the marked fall in the death-rate from phthisis.

(c) *Treatment by tuberculin* depends upon the principle of immunisation (Chapter XVIII.). Koch followed his discovery of the tubercle bacillus by soon afterwards issuing to the world the toxin produced by the bacillus. This he called tuberculin, and it is now called the *old* tuberculin. It was administered hypodermically and produced considerable "reaction," *i.e.*, constitutional disturbance, in the patient. It was largely used, especially in cases of lupus;<sup>1</sup> but it is now admitted on all hands to be useless as a remedial agent, though it is useful for purposes of diagnosis, because the reaction can only be obtained in persons suffering from tubercle in some part of their body.<sup>2</sup> It is chiefly used in Germany. Since then, however, in the year 1897, Koch issued another preparation known now as the *new* tuberculin, which is undoubtedly an efficacious remedy (see serumtherapy).

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<sup>1</sup> Tuberculosis of the Skin.

<sup>2</sup> Even as a diagnostic test the old tuberculin is not free from danger, *viz.*, the dissemination of the disease to other parts of the body. Dr. George Dean states that whilst he was working at Vienna several cases of children with chronic tuberculous bone disease died of acute miliary tuberculosis within a few months of inoculation with the old tuberculin.

(d) The “*open-air*,” hygienic, or sanatorium treatment of phthisis as it is now called, is not altogether a new method, for fresh air has always been advocated as advantageous to these patients. But the systematic open-air treatment has only been in vogue within the last few years, as in Germany at Nordrach, and latterly in England ; and there are now numerous sanatoria devoted entirely to this plan of treatment.<sup>1</sup> Much discussion has taken place as to whether it cannot be carried out without a sanatorium.<sup>2</sup> Among the well-to-do perhaps a sanatorium is not indispensable, but in the middle and lower classes the necessary discipline cannot be otherwise carried out. That residence in a sanatorium is not absolutely necessary is evidenced by cases which have been under my care, even in an advanced stage of phthisis, who were unable to go away. One of them spent all the daytime in Kensington Gardens, in all weathers, and when indoors the windows were always open. One recovered in six months. See also a case reported in the *Lancet*, Jan. 20, 1900.

Briefly, the *advantages* gained by this method of treatment consist of:—(i.) Increased medical supervision from day to day, and hour to hour, by the medical officer of the sanatorium ; (ii.) the continuous exposure of the patient to fresh, pure air, night and day, the windows never being shut, and sometimes wholly removed ; (iii.) systematic exercise in suitable cases ; (iv.) the ingestion of a large amount of suitable food ; (v.) a suitable amount of rest during the fever stage, and a freedom from excitement ; (vi.) the avoidance of mixed infections, by hygienic mode of life. Cleanliness and fresh air tend to obviate pyogenic processes and infections. All possibility of the introduction of influenza and other infective disorders should be obviated by the *proper regulation of visitors* to patients. I believe that some day these latter will be subjected to the most rigorous scrutiny and inquiry before being allowed to come in contact with the consumptive patients in a sanatorium. The mixed or superadded infections do more harm than the tubercle bacillus. This is probably the reason why tuberculous patients do so badly in the wards of a general hospital.

<sup>1</sup> Lists of sanatoria are given in the *Lancet*, 1899, ii. pp. 724, 851.

<sup>2</sup> Discussion, Roy. Med. Chir. Soc., Dec. 1899.

The possible *disadvantages* urged are :—(i.) The fear of hyper-medication that may go on in sanatoria, and (ii.) certain cases attended with a good deal of bronchial catarrh are said to be deleteriously affected.

In carrying out this treatment, seven rules should be observed :—

(1) Much depends on the suitability of the case, and the *earlier the stage* the better. There are three conditions in which the sanatorium, or open-air treatment, is undesirable :—(i.) When the process is too active, as evidenced by a high and wide range of temperature ;<sup>1</sup> (ii.) when the lungs are too far destroyed, and (iii.) when the case is attended by active bronchial catarrh.

(2) The food must be very abundant, and the cuisine appetising and attractive.<sup>2</sup> But here an important caution comes in, else the patient puts on fat without influencing the disease. The food must be in proportion to the exercise, and the patient's weight should never much exceed what his previously normal weight was. The proteid foods should be increased relatively to the farinaceous, otherwise the patient becomes plethoric and breathless.

(3) Evidences of benefit should be carefully looked for, and they are three in number : (i.) a lowering of the temperature, and a lessening of its range ; (ii.) an increase in the appetite ; (iii.) increase of weight *combined* with the two previous features. This should be combined with the caution above mentioned.

(4) In deciding the important question of rest or exercise the great value of accurate temperature records is again seen. When the highest daily record exceeds 102° the patient should be confined to bed. If the highest record exceeds 100·5° he should only be allowed to move about 100 yards at a time. If it does not exceed 100·5° then daily out-door exercise should be enforced, and especially slow up-hill walking, for this strengthens the heart, expands the lungs, and improves the tone of the body generally.

(5) Amusement is necessary, but it requires to be carefully regulated. The patient should not talk too much, and any excitement or heated discussion is bad. *The whole day, and if possible*

<sup>1</sup> Some do not regard this as a contra-indication to open-air treatment ; but in such cases the length of the journey has to be considered, and the undesirability of mixing such patients with others.

<sup>2</sup> Thirteen pints of milk or its equivalent is in most institutions adopted as a fair standard of diet ; that is, 9 oz. proteid, 7½ oz. fat, 10½ oz. carbohydrate—total, 27½ oz. water-free food. With "Parkes' Hygiene" percentage composition tables varying diets can be easily made up, containing the above proportion of food elements.

the night also, should be spent out of doors, no matter what the weather may be, and out-door amusement cultivated. A very useful contrivance is the Liegehalle, which is practically a small revolving summer-house, the front of which is open, and can be turned away from the wind.

(6) The duration of the treatment must be sufficient, and should be continued for some time after all symptoms have disappeared.<sup>1</sup>

(7) The hygiene and the locality of the building are important matters, but the reader must refer to special works for this.

§ 98a. **Fibroid Phthisis** is one of the least common of the varieties of pulmonary tuberculosis. It may be defined as a tuberculo-fibroid disease of the lungs, occurring for the most part in elderly subjects, running a protracted course, and terminating in contraction of the lung. This disease is very apt to be confused with chronic interstitial pneumonia or cirrhosis of the lung (§ 101).<sup>2</sup>

*Symptoms.* The disease is essentially one of insidious onset and long duration. The patient complains of a chronic cough for many years. Later on this may become paroxysmal, and especially troublesome in the morning. Progressive shortness of breath, clubbed fingers, slowly increasing weakness and emaciation, with little or no fever, constitute the other symptoms.

The *physical signs* begin and are almost always most marked at the apex. *Both lungs* are usually affected (which contrasts with interstitial pneumonia), but the signs of disease are afterwards more advanced on one side. There is impairment of the chest movement, and later on contraction of one side of the chest. The area of præcordial dulness is increased when the left lung is involved; and the heart and other viscera may be displaced. The signs of consolidation, with gradual softening, may also be present. Hamoptysis sometimes occurs, and the tubercle bacillus may be discovered on careful and repeated examination of the sputum.

The *diagnosis* from other forms of *phthisis* is made by the extremely protracted course of this disease and the age of the patient. *Chronic interstitial pneumonia* resembles it very closely both in its physical signs and symptoms, and the diagnosis of interstitial pneumonia can only be inferred (i.) from the absence of the tubercle bacillus after oft-repeated examinations, and (ii.) from the more usual localisation in one lung.

*Etiology.* Fibroid phthisis is more frequently met with at and after middle life. It may follow chronic bronchitis, broncho-pneumonia, or repeated attacks of pleurisy. In true Fibroid Phthisis the tubercle bacillus is primarily deposited in a healthy lung under the same circumstances as in chronic pulmonary tuberculosis, and then takes on an indolent fibroid growth. On the other hand, chronic interstitial

<sup>1</sup> According to Clifford Allbutt (*Disen. on Tuberculosis*, *B. M. J.*, Oct. 28, 1899):—If the case is only in the first stage cure may be accomplished in six months; if the second stage is reached (consolidation) cure can only be accomplished after a disciplinary treatment of eighteen months; and in the third stage cure is rare, but is possible if the treatment be carried over two or three years.

<sup>2</sup> Reference to chronic interstitial pneumonia (§ 101) will show to what condition the term Fibroid Phthisis should be confined.



pneumonia may become the seat of tuberculous invasion, and in that case the causes of chronic interstitial pneumonia are the causes of fibroid phthisis (see interstitial pneumonia, § 101).

*Prognosis.* Its course is extremely indefinite, but sooner or later it terminates fatally. The chief complications are bronchiectasis, compensatory emphysema of the lungs, lardaceous disease of other organs, and cardiac failure. In general terms the prognosis depends upon the same conditions as those of pulmonary tuberculosis, and the *treatment* is the same.

II. *The patient complains of breathlessness; on examining the chest dulness is found at one or both bases, and SIGNS OF FLUID are detected there—the disease is HYDROTHORAX.*

§ 99. **Hydrothorax** is a chronic collection of serous fluid in the pleural cavity, differing from the effusion of pleurisy in being non-inflammatory. The surfaces of the pleuræ are smooth, the fluid is free from flakes, and generally alters its level with the position of the patient.

*Symptoms.* The general symptoms may be but little marked if the fluid is small in quantity. The onset is usually gradual. Dyspnoea is generally present, especially on exercise, but its degree depends upon the amount of fluid. As hydrothorax is always a secondary condition, the symptoms may be masked by the presence of dropsy elsewhere; and it is remarkable how often hydrothorax is overlooked on this account. In rare cases the fluid collects with great rapidity, as in a fatal case of double hydrothorax supervening on scarlatina, mentioned by Osler.<sup>1</sup>

The *physical signs* are those of fluid in the chest (*vide* § 80). The level of the fluid in hydrothorax, unless excessive in quantity, moves when the patient alters his position, thus differing from the inflammatory fluid of acute pleurisy. This is an important diagnostic feature which can always be elicited, except where the fluid is confined by adhesions.

*Diagnosis.* The disease has to be diagnosed from other disorders giving rise to dulness on percussion (p. 199). As regards *pleurisy*, in addition to the mobility of the fluid, hydrothorax is distinguished by the absence of pyrexia at the onset, by the absence of pain, and by the fact that the fluid occurs usually on both sides. The use of the aspirating needle settles the diagnosis from *consolidation* (see also Table, p. 169).

<sup>1</sup> Osler, "Prin. and Pract. of Medicine," 2nd ed., p. 608.

*Etiology.* (i.) Hydrothorax may form part of the *general* dropsy of Bright's disease, in which circumstances both pleuræ are involved. Here the hydrothorax is of no very great importance *per se*, but the onset of dyspnœa in Bright's disease should always direct our attention to the pleuræ. (ii.) Similarly it may form part of *cardiac* dropsy, in which circumstances one pleura is generally solely or chiefly affected. (iii.) New growths in the pleura are generally attended by hydrothorax. This is especially so in the case of carcinoma, which should always be suspected in the aged. In this case the fluid is blood-stained and may be found to contain cancer cells. In tubercle there is rarely much fluid in the pleura, adhesions being more common. (iv.) Aneurysm or other intra-thoracic tumours pressing on the veins of the thorax may give rise to hydrothorax on one or both sides.

*Prognosis.* The disease is essentially chronic, and runs a prolonged and indefinite course, the duration depending very much upon the cause. In general terms the prognosis of the condition is favourable, except in cases of new growths. The patient should be carefully watched for the occurrence of shivering, sweating, or intermitting pyrexia, as indicative of empyema. The sudden onset of signs of fluid in the chest, accompanied by shock or collapse in a case which has previously presented the symptoms of aneurysm, points to the occurrence of hæmorrhage into the pleural cavity.

*Treatment.* The treatment is comparatively simple. The administration of brisk hydragogue purgatives will generally reduce the amount of fluid; if this fails, or if the fluid return, or in any case where dyspnœa is extreme, paracentesis (p. 182) should be resorted to. The operation of tapping may be repeated indefinitely. Diuretics or cardiac stimulants are useful. For the rest, the treatment must be directed to the primary condition. See also § 89.

III. *The patient complains of breathlessness; on examining the chest dulness is found at one or both bases; and on auscultation, FINE CREPITATIONS are heard—the disease is PULMONARY CONGESTION OR ŒDEMA.*

§ 100. **Œdema of the lung** (Pulmonary congestion). Œdema of the lung is a serous exudation into and around the

air vesicles. It is synonymous with the term hypostatic congestion, or, as it is sometimes called, hypostatic pneumonia. It constitutes the termination of many serious disorders.

*Symptoms.* (i.) It is practically never a primary condition, and therefore our attention is first directed to the symptoms of its cause. The advent of hypostatic congestion is always insidious, and it is only by careful watching that its onset can be detected. (ii.) A considerable amount of dyspnoea is present, which may amount to orthopnoea. (iii.) There is an abundant frothy mucous expectoration, not infrequently tinged with blood.

The *physical signs* are somewhat indefinite, but they are found, as is implied by the term "hypostatic," chiefly at the bases of both lungs. The percussion note is somewhat impaired, and the R.M. at the bases is diminished, and is attended by abundant moist crepitations.

*Diagnosis.* The condition is diagnosed from true pneumonia by the gradual onset, the indefinite signs, and the absence, for the most part, of pyrexia and other constitutional symptoms. Any rise of temperature that may be present is due to the primary or causal condition.

*Etiology.* (i.) The disease is most frequently met with in elderly people. (ii.) Pulmonary œdema complicates various blood disorders and fevers, especially typhus and typhoid fevers. The latter indeed is so frequently complicated in this way that hypostatic congestion is an aid to the diagnosis in the second and third weeks of the disease. In Bright's disease and anæmic disorders, œdema of the lungs appears as part of a generalised dropsy. (iii.) Cardiac diseases, and other disorders leading to mechanical dropsy, produce œdema of the lungs. (iv.) Tumours pressing on the veins within the mediastinum may result in pulmonary œdema.

*Prognosis.* The prognosis is always grave because pulmonary œdema indicates considerable impediment to the circulation in the lungs, or a serious toxic condition of the blood. It frequently terminates life in circulatory disorders, and in specific fevers of the asthenic type. In pneumonia it heralds a fatal issue. Œdema of the lungs is one of the most constant lesions found in the

typhoid state. As regards untoward symptoms, the extent of the œdema is indicated very fairly by the degree of dyspnœa.

*Treatment.* The indications are to relieve the cause, if possible, and to stimulate the heart. Ammon. carb. and other stimulating expectorants aid the heart, and promote expectoration. The liberal administration of alcohol and other diffusible stimulants is called for. In the aged, among whom even slight disorders are apt to be attended by pulmonary œdema, it is well to *keep the patient propped up* in a semi-recumbent posture. For the same reason it is advisable in cases of fracture and other surgical maladies in the aged, to get them up as soon as possible, even at the risk of doing harm to their surgical ailment, so as to obviate the occurrence of hypostatic congestion.

GROUP b. *We now turn to the rarer chronic diseases attended by dulness on percussion; in which the dulness occurs in irregular and scattered areas:—*IV. INTERSTITIAL PNEUMONIA; V. THICKENED PLEURA; VI. CANCER AND OTHER TUMOURS; VII. COLLAPSE; VIII. SYPHILITIC DISEASE; and IX. MEDIASTINAL TUMOURS.

§ 101. **Chronic Interstitial Pneumonia**—apart from that form due to the malign effects of certain trades—is a rare disease. It may be defined as a Chronic Interstitial Fibrosis of the lung, localised or diffuse in the different varieties, running a protracted course, and resulting in contraction of the pulmonary tissue.

An increase of the fibrous tissue of the lung may take place under the following circumstances, all being chronic processes:—

(i.) An indolent growing tuberculous deposit may assume a fibroid character. Fibrosis is one of the ordinary terminations of a tuberculous deposit; but when the process is very slow and protracted, with excessive formation of fibrous tissue, it constitutes true *Fibroid Phthisis*.

(ii.) The constant inhalation of dust in certain trades (*e.g.*, fustian cutters, jute workers, wool sorters, stone, knife, and other grinders and polishers, iron and coal miners, etc.) gives rise to a *chronic broncho-pneumonia*, followed by a peri-bronchial fibrosis, which later on involves considerable areas of lung tissue.

(iii.) Repeated attacks of *pleurisy* may be attended by a sub-pleural fibrosis (thickened pleura), and dense bands of fibrous tissue may extend into the lung (Sir Andrew Clark).

(iv.) *Acute broncho-pneumonia* becoming chronic may, although very rarely, result in an interstitial fibrosis. This form nearly always terminates by becoming tuberculous.

(v.) An *acute lobar pneumonia* similarly may assume a chronic course, and may result in an interstitial fibrosis (Addison). This form has not the same tendency to become tuberculous.



(vi.) *Syphilitic disease* of the lung is very rare, excepting as an hereditary manifestation in infancy, in which circumstances the change consists of a fibroid induration of the lung (Kingston Fowler).

All of these may become the seat of tuberculous disease, but only the first, which is a *tuberculo-fibroid* process, should be called Fibroid Phthisis. The other varieties constitute Cirrhosis of the lung; and if they are invaded by the tubercle bacillus they form a *fibro-tuberculous* process, which in its later stages may be indistinguishable from Fibroid Phthisis.

The general *symptoms* consist of progressive weakness and dyspnoea. There is no fever unless there is ulceration of the bronchi or septicæmia—a common occurrence in late stages of the disease.

The physical *signs* may be found either at the base or the apex, though usually the former. Except in the variety due to the inhalation of irritating particles, only *one lung* is involved, thus differing from fibroid phthisis, in which both lungs are usually affected. There is deficient mobility of the diseased side, and later on it undergoes contraction, so that there may be considerable difference in the measurement of the two sides of the chest. There is dulness on percussion; on auscultation bubbling râles may be heard, but sometimes the only symptom is weak bronchial breathing or a weak respiratory murmur. The expectoration sometimes contains blood, but never the tubercle bacillus.

The *diagnosis* of Interstitial Pneumonia from Fibroid Phthisis is sometimes very difficult, as may be seen in the description of the various processes just named. It is also liable to be mistaken for empyema.

The *etiology* of the condition is given above. It is met with chiefly in male subjects under the age of 50—especially between 15 and 30. Alcoholism predisposes. The commonest form of chronic interstitial pneumonia is that met with in persons engaged in trades attended by the inhalation of irritating particles. Sometimes it is a sequel to other pulmonary disorders.

*Prognosis.* The prognosis is serious, because nothing will remove the fibrous tissue. As regards the duration of life, the prognosis is good if the patient is not losing weight and the disease is not too far advanced. The *complications* are bronchiectasis, a very frequent sequel, dilated right heart, and emphysema occurring in other parts of the lung. When ulceration of the bronchi has taken place, lardaceous disease and septicæmia may ensue.

*Treatment.* Counter-irritation and respiratory exercises are given on the lines advised in chronic phthisis (§ 98). F. 61 is useful.

**§ 102. Thickened Pleura** is a condition which sometimes succeeds dry pleurisy, especially recurrent dry pleurisy. It is important to be able to recognise it, lest it should be mistaken for some more serious condition, though it is somewhat difficult to do so. It is more often localised to one part, and the commonest thickening of the pleura is that at the apex associated with chronic phthisis.

The *symptoms* of thickened pleura are:—(i.) a localised enfeeblement of the respiratory murmur; (ii.) a dulness on percussion; and (iii.) a slight diminution in the V.R. and the V.F.

The *diagnosis* is arrived at (i.) by the history of the case, *e.g.*, there has been an attack of pleurisy or pneumonia in the past; and (ii.) by the absence of signs of active disease when the patient is kept for some time

under observation. The condition is often discovered only by chance, when the patient seeks advice for other ailments.

*Treatment* is of no avail; and if only moderate in degree the disease is not of much consequence. Counter-irritation may be applied.

§ 103. **Malignant Disease of the Lung.** Cancer of the lung is rarely or never a primary condition; it is most frequently secondary to cancer of the breast, liver, or spleen. It occurs in two forms—(a) hard, nodular, isolated masses, when it is probably scirrhous or epithelioma; (b) a soft, irregular infiltration, probably encephaloid cancer. The evidences of the former are usually more distinct than those of the latter.

*Symptoms.* The lung trouble may be preceded by signs of malignant disease elsewhere. The first evidence of involvement of the lung is breathlessness. This is followed by cough, and by expectoration which may from time to time be tinged with blood. Pain is often present, and indicates generally that the pleura is invaded, in which case there is usually a certain amount of pleuritic (blood-stained) effusion.

The *physical signs* are often very indefinite. Clinically there are two forms—(a) the *nodular* form is usually attended by serous effusion (see hydrothorax, § 99). Effusion into the pleura coming on slowly, or returning persistently, in an elderly person is of itself suspicious, and the diagnosis is confirmed when on aspiration the fluid is found to be blood-stained. Sometimes in the midst of what appears to be a hydrothorax, we detect the signs of consolidation; this probably indicates that the neoplasm has come to the surface in one locality.

(b) With the *infiltrated* form, we find signs of consolidation accompanied later on by the moist sounds due to the breaking down of the growth. Here again nearly every variety of physical sign may be met with in different parts of the lung, and if the main bronchus be obstructed there is entire absence of the R.M.

*Diagnosis.* The condition has to be diagnosed from different forms of pneumonia, and from pleurisy with effusion, and hydrothorax. The age of the patient, the chronic course of the affection, the absence (usually) of pyrexia, and the presence of cachexia, should enable us to come to a conclusion.

*Prognosis.* The question is one of duration, and this can only be gauged by daily observation of the case, and by noting the rate at which the growth appears to be spreading. Death usually occurs in about six months.

The *treatment* resolves itself into the relief of pain and the amelioration of other symptoms which may be present.

§ 104. **Atelectasis, or collapse** of the lung, is a condition in which the lung tissue is not expanded. The term *Atelectasis* is usually applied to lung tissue which has never properly expanded, and is therefore a congenital condition, due to imperfect development. The term *Collapse of the Lung* is applied to lung tissue which has previously expanded, but in which the air vesicles have subsequently collapsed.

**Atelectasis** is a *congenital* condition, and the symptoms occur in the new-born child, and consist of cyanosis, with shallow, rapid respiration. The lower part of the chest is drawn in by each respiration. On auscultation the R.M. is found to be very faint.

The symptoms of **collapse of the lung** follow and complicate those of the disease which has led to the condition: for instance, the patient may

not recover so rapidly as he ought, or the breathing is more embarrassed than can be accounted for by the concurrent disease in the chest. The physical signs vary considerably with the degree of collapse. Thus:—

(a) In *complete* collapse of a part of the lung, as, for instance, in collapse due to compression or complete obstruction of a bronchus high up, there is impairment of the percussion note, a diminution or absence of the R.M., of the V.R., and of the V.F.

(b) Where the collapse is only *partial* in degree, *e.g.*, where the bronchi remain patent, as occurs sometimes when the lung is compressed by pleuritic or pericardial effusion, there are signs resembling those of consolidation (p. 169), except that the percussion dulness is not so marked, and the R.M., though bronchial in character, is somewhat feeble.

(c) Where the collapse is *slight* and limited, the chief sign is an enfeebled R.M. In addition, during deep inspiration are heard fine rustling crepitations, due to the expansion of the collapsed vesicles.

The *diagnosis* is made usually by the existence of a causal condition. When this is detected, attention may then be directed to the physical signs of the lungs. It will be observed that the signs of partial collapse resemble the signs of consolidation, and those due to slight collapse resemble early pneumonia.

*Etiology.* The causes are of two kinds: (a) Causes which produce *obstruction*, such as (i.) a tumour at the root of the lung (*e.g.*, aneurysm); (ii.) obstruction in the throat (? adenoids); (iii.) stricture of a bronchus (*e.g.*, gumma); (iv.) secretion obstructing the bronchi, though this is only sufficient to cause obstruction in children, suffering for example from measles, whooping-cough, or broncho-pneumonia; (v.) foreign bodies obstructing the larynx or bronchus.

(b) *Compression* of the lung may be produced by pleural or pericardial effusion, an enlarged heart, tumours of the mediastinum, or wounds of the chest-wall. The condition is often the result of spinal curvature.

In *adults* collapse is most often met with as the result of pleural effusion or tumours in the chest; in *children*, of bronchitis or broncho-pneumonia.

*Prognosis.* The course of the disease depends very much upon the cause. Recovery soon takes place after compression by effusions, obstruction or stricture of the bronchi, and throat affections. It is unfavourable when associated with mediastinal tumours or foreign bodies in the bronchus.

The *treatment* is unsatisfactory. It should be directed to the removal of the cause, and especially to the promotion of recovery of any concurrent pulmonary disorder. That form which yields best to treatment is met with in children with bronchitis and broncho-pneumonia. In adults it might be well to try the efficacy of respiratory exercises.<sup>1</sup>

§ 105. **Syphilis of the Lung.** Syphilitic disease of the lung may take one of two forms: (a) The pneumonic condition of lung which is found in infants, usually still-born, is universally regarded as an interstitial pneumonia of syphilitic origin.

(b) *Gummata* are occasionally met with in the lungs of infants who are the subjects of hereditary syphilis; still more rarely, they are met with in adults.

Dyspnoea is usually the only symptom. The signs are those of consolidation, and collapse.

In adults syphilis of the lung may take other forms, *e.g.*, broncho-pneumonia, bronchiectasis, etc., and may lead to extensive infiltration and breaking down, or to cirrhosis.

<sup>1</sup> "Respiratory Exercises in Treatment of Disease notably of the Heart, Lungs, Nervous, and Digestive Systems," by Dr. Harry Campbell. London, 1899.

GROUP C. CHRONIC DISEASES attended by **Hyper-resonance** on percussion. I. In quite nine cases of hyper-resonance out of ten it exists on both sides and is due to EMPHYSEMA. There are five other conditions which give rise to it, namely: II. PNEUMOTHORAX (§ 93); III. SKODAIK RESONANCE (§ 78); IV. A very large CAVITY in the lung (Phthisis, § 98); V. A TUMOUR between the chest-wall and a large bronchus (§ 103); VI. DISSEMINATION OF SOLID MATERIAL through the lung in certain exceptional circumstances (*e.g.*, Pneumonia, Sarcoma, etc.); the diagnosis of these various conditions is given in the form of a table. All except EMPHYSEMA are described elsewhere.

TABLE XI.—CAUSES OF HYPER-RESONANCE.

Cause.	Hyper-resonance.	Auscultation.	Other Diagnostic Features.
I. <b>Emphysema.</b>	Bilateral and universal.	R.M. distinct and expn. much prolonged; signs of bronchitis, if present.	Barrel-shaped chest, cardiac dullness obscured, and organs displaced.
II. <b>Pneumothorax</b> , mostly hydro-pneumothorax. An acute condn.	Hyper-resonance always unilateral, though it may extend beyond middle line.	Absence of R.M. and V.F. over affected area; sometimes amphoric breathing. Bell-sound.	Organs displaced; history of emphysema or tubercular cavity.
III. <b>Skodaic Resonance</b> , <i>i.e.</i> , the high-pitched note above a large pleuritic effusion, when the lung is otherwise healthy.	Unilateral; level may shift with position of patient.	Loud R.M.; V.F. felt over affected area.	History of pleurisy; signs of fluid lower part of chest.
IV. A very large cavity, or extensive bronchiectasis (rare).	Unilateral, and of limited extent (may be cracked-pot sound).	Amphoric breathing, whispering pectoriloquy.	Expectoration of pus and long history of phthisis or bronchitis.
V. A <b>Tumour</b> (or pneumonic consolidation) between the chest-wall and a large bronchus (rare).	Unilateral, and of limited extent; dullness elsewhere.	Tubular breathing and bronchophony	Symptoms of intra-thoracic tumour.
VI. <b>Infiltration of solid</b> and even fluid material through the lung, <i>e.g.</i> , early stage of pneumonia, miliary tubercle, etc. (rare).	Hyper-resonance not marked (may be bilateral).	Signs of consolidation in some parts.	Hyper-resonance generally transient.



I. *The patient has complained of breathlessness for some years. There is Hyper-resonance on both sides of the chest—the disease is EMPHYSEMA.*

§ 106. **Emphysema** is a chronic non-febrile disease of the lungs in which the air vesicles become hyper-distended, the walls separating each vesicle become atrophied, inelastic and ruptured, and as a result the aerating surface is greatly diminished and the lungs deficient in their elastic recoil.

*Symptoms.* (1) The onset of the disease is imperceptible and generally supervenes gradually after repeated attacks of bronchitis, the patient becoming more and more breathless after each attack. (2) This breathlessness is practically the only symptom, and it differs from all other kinds of breathlessness in this, that the chest remains *permanently in the inspiratory position*—in other words, owing to the inelastic state of the lungs and the shape of the chest, the patient finds it more difficult to expire than to inspire. A certain degree of cyanosis is generally present. (3) Symptoms of bronchitis are *nearly always present*.

The *physical signs*, expressed *briefly*, are a barrel-shaped chest, hyper-resonance and prolonged expiration. The shape of the chest is special to emphysema (see Fig. 36). Tersely put, the chest assumes permanently the shape of a healthy chest in a position of deep inspiration. The antero-posterior diameter is considerably increased (see § 77). The hyper-resonance is always bilateral, and it obscures the dulness of the neighbouring organs, namely, the heart, the liver, and the spleen. These organs are also displaced downwards. The apex-beat may not be palpable, but epigastric pulsation is usually felt. On auscultation, the respiratory murmur is modified; the inspiratory sound, which is full, is followed by a pause and then by a prolonged expiratory sound. There are no adventitious sounds proper to emphysema, but, as just mentioned, bronchitis (*q. v.*) nearly always accompanies it. Well-established emphysema interferes considerably with the pulmonary circulation, on account of the ruptured alveoli, and consequently the right side of the heart in course of time becomes dilated.

*Variety.* In old people there is sometimes hyper-resonance with a weak R. M., but no enlarged barrel chest; this is called

*Atrophic Emphysema*, and is due to the giving way of degenerate air vesicles.

The *diagnosis* is extremely easy, because the bilateral hyper-resonance, the prolonged expiration, and the barrel-shaped chest are quite characteristic (*vide* table of diagnosis).

*Etiology.* (i.) Emphysema occurs usually in elderly subjects. Both sexes are affected, but it is much commoner in males owing to the prevalence of bronchitis and asthma in them. (ii.) Heredity is said to play no part in the disease, but undoubtedly a hereditary tendency can frequently be traced. (iii.) Certain occupations render people prone to emphysema, *i.e.*, those which throw strain upon the lungs, such as glass-blowers, wind-instrument blowers, etc. (iv.) The disease is frequently associated with senile degeneration, chronic Bright's disease, and cardio-vascular changes. (v.) Bronchitis is the most frequent of the exciting causes, owing to the prolonged coughing and straining to get up phlegm, and owing also to the blocking of certain tubes with thickened secretion, which prevents the access of air to some alveoli and unduly distends others. (vi.) Asthma is also a potent exciting cause, owing to the constant strain on the elastic tissue of the lungs.

*Prognosis.* Patients may live with emphysema to a good old age, and provided it is only moderate in degree it does not necessarily shorten life, though it predisposes to and adds to the seriousness of other pulmonary disorders. The gravity of any particular case is best measured by the extent of cardiac involvement (*q. v.*).

*Treatment.* The indications are:—(i.) to relieve the accompanying bronchitis (see § 97); (ii.) to improve the cardiac condition; and (iii.) to restore as far as may be the elasticity of the lungs. The diet is of considerable importance in advanced emphysema, for any distension of the stomach greatly adds to the respiratory distress. It is a good rule never to let patients take a solid meal later than two o'clock in the day, otherwise their nights become considerably disturbed by the breathlessness. Cardiac tonics, especially strychnine and, in my experience, *tinctura cacti grandiflori*, are useful. Quinine and cod-liver oil often do good, although I cannot explain how the latter acts.

To restore the elasticity of the lungs is important, but difficult

to accomplish satisfactorily; but of late years a special form of respiratory exercise has been put forward as fulfilling this condition and to relieve the difficulty of expiration. With this end in view Gerrhardt<sup>1</sup> has recommended the employment of mechanical expiration by compression of the thorax methodically, every day for five or ten minutes, by another person who places his two hands flat upon either side of the patient's chest. A similar result has been attained by Rossbach's althemstühl (breathing-chair).

GROUP d. There are three chronic pulmonary conditions in which the percussion note varies considerably in different cases, but the **offensive character of the sputum** reveals their presence, viz., I. BRONCHIECTASIS, II. GANGRENE, and III. ABSCESS of the lung. In Abscess the sputum is not so invariably offensive as in the others.

§ 107. **Bronchiectasis.** Bronchiectasis is a cylindrical or saccular dilatation of the bronchial tubes. The condition is met with most frequently as a complication of chronic bronchitis or chronic pneumonia.

*Symptoms.* The patient complains chiefly of persistent cough. At intervals of several days violent increase of coughing occurs; it is started perhaps by some change of posture, and is followed by the expectoration of a large quantity of extremely fœtid sputum. In the intervals the sputum is scanty, but the breath is offensive. The fœtid sputum contains pellets or "Traube plugs," and sets characteristically in three layers (see § 82).

The *physical signs*, if present, are mostly those of a cavity, attended by general signs of chronic bronchitis in both lungs; occasionally only one is involved. The patient is often cyanosed, and has clubbed fingers.

*Diagnosis.* The extremely fœtid odour of the sputum—occurring as it does at intervals of perhaps several days or weeks in which the sputum is not fœtid—distinguishes bronchiectasis from all other diseases. In *Gangrene* of the lung the sputum may be fœtid, although in a less degree, and it lacks the intermittent character. The position of the bronchiectasis is generally marked by dulness in the lower lobe of one lung, which may be made to disappear by making the patient lie face downwards for some time with his head low, till he coughs up a large amount of sputum. The causes of the two affections also aid the diagnosis. Abscess of the lung is attended by a very profuse purulent expectoration, but it is not so fœtid.

*Etiology.* (i.) By far the most usual cause is prolonged chronic bronchitis occurring in patients past middle life. The dilated bronchial tube results from the continual strain of coughing on the weakened walls. (ii.) Various forms of chronic pneumonia and chronic phthisis are believed to be attended by bronchiectasis, but in such cases it is probably a cavity in the lung tissue and not true bronchiectasis that we meet with. (iii.) A foreign body plugging a bronchus is an occasional cause. (iv.) In very rare cases it is a congenital defect.

<sup>1</sup> Strumpel's "Pathologie und Therapie."

*Prognosis.* The condition is a very serious one, and for the most part incurable; the patient may live from one to ten years. The prognosis is much worse in bilateral cases or in cases associated with extensive disease of the lungs or pleura.

The *complications* which may occur are, fatal hæmorrhage, gangrene of the lung, lobular pneumonia, and pyæmia.

*Treatment.* The indications are to relieve the disgusting fœtor and to cure the primary disease. The first is accomplished by liberal antiseptic inhalations, of turpentine, coal-tar, creosote.<sup>1</sup> Terebene and creosote are almost specifics. They may be given in capsules (4 minims) three times a day. Some inject menthol or guaiacol into the trachea in the proportion of 5 and 2 grs. in 3j olive oil twice a day. When the cavity is low down surgical measures for its drainage have been adopted.

**§ 108. Gangrene of the Lung.**—Owing to the extreme vascularity of the pulmonary tissues, gangrene of the lung is a rare condition, but it occasionally occurs over a limited area. It is usually a secondary condition, but it sometimes occurs in a lung previously healthy.

*Symptoms.* 1. The onset may be sudden, and marked by prostration and an irregular intermittent pyrexia of a pyæmic type with a very rapid pulse. 2. If, as is usual, the gangrenous part opens into the bronchi, we soon get a profuse fœtid expectoration. The sputum contains fragments of lung tissue, and generally blood also. The fœtor is continuous, and not intermittent as in bronchiectasis. 3. The breath is extremely fetid. 4. Pain in the side is usual, though it depends upon the involvement of the pleura. There is persistent cough which aggravates the pain. 5. The *physical signs* are those of consolidation, sometimes those of a cavity.

*Diagnosis.* The only condition which is liable to be mistaken for it, by reason of its fetid expectoration, is bronchiectasis, which is distinguished by having (i.) "Traube" plugs in the sputum, and (ii.) a gradual onset, and longer course.

*Etiology.* It is predisposed to by intemperance, old age, diabetes and marasmus. Exciting causes are:—(i.) Particles of food entering the lung, as in the insane, or patients with laryngeal paralysis, or persons in a drunken coma. In children a foreign body swallowed may produce it, though rarely. (ii.) Septic matter passing from the throat or mouth. (iii.) Severe sthenic types of pneumonia are occasionally so complicated. (iv.) It may complicate bronchiectasis. (v.) Embolism of the pulmonary artery. (vi.) Aneurysm pressing on the root of the lung. (vii.) The extension of an abscess near the lung.

*Prognosis.* The disease is almost invariably fatal either immediately, from collapse, sometimes from fatal hæmorrhage, or later from prostration. A few cases have recovered where the patch was of small extent. In cases which have been recorded as lasting for months or years it is very doubtful whether the lesion was true gangrene; bronchiectasis is more probable. Occasionally the condition leads to pyo-pneumothorax.

*Treatment.* The main object is to keep up the strength of the patient by means of abundant nutritive stimulants, bark, iron and quinine. For the rest the treatment is the same as in bronchiectasis.

**§ 108a. Abscess of the Lung** is a serious and happily rare condition, but as it is nearly always secondary to some grave and often fatal disorder, it does not add very materially to the gravity of the situation.

It is usually manifested by the expectoration of a large quantity of purulent pus which may or may not be fœtid, and is never so fœtid as in bronchiectasis or gangrene. The constitutional disturbance to which it gives rise is usually marked by that of the primary malady. The physical signs are those of localised consolidation, but these also are generally masked by those of the primary lesion.

It may occur in the course of: (i.) Advanced pulmonary tuberculosis; (ii.) pneumonia; (iii.) pyæmia; (iv.) cancer or other tumours of the lung, such as gummatæ breaking down, a somewhat rare condition; (v.) it occasionally follows the introduction of septic foreign bodies or wounds of the throat.

*Prognosis.* The prognosis is very grave, but depends upon the causes. Occurring in the course of pyæmia it indicates the progress towards a fatal termination. It is less grave in pneumonia occurring in otherwise healthy persons.

*Treatment.* Medical treatment is not of much use. Surgical interference is not good in malignant and pyæmic conditions, but in other conditions, if fairly superficial, the abscess may be drained.



## CHAPTER VII.

### THE UPPER RESPIRATORY PASSAGES ; AND THE THYROID GLAND.

THE Throat, or "swallow," may be the seat of the same morbid processes as affect other mucous structures, viz., catarrh, ulceration, new growths, &c. It is, moreover, in this position that several very important general or constitutional maladies, such as diphtheria, scarlatina, and syphilis, have their chief local manifestations. These facts have long been known, but only quite recently it has come to be recognised that the throat, and especially the tonsils—organs whose functions are still imperfectly known—may constitute the portal of entry of certain microbic conditions. It has also been suggested that the virus of influenza, rheumatism, malignant endocarditis, and other septic conditions thus gain admission into the general systemic circulation.

This chapter will deal with the symptoms referable to the **pharynx** (p. 225), the **larynx** (p. 237), the **nasal cavities** (p. 252), and the **thyroid gland** (p. 265).

#### The Throat.

§ 109. *SYMPTOMATOLOGY*.—"The throat" may be said to consist of the fauces, tonsils, palate, pharynx, and larynx ; and we are here concerned with the investigation of these structures. The symptoms indicating disease of these parts are principally two, namely, SORE THROAT and HOARSENESS. The examination of the mouth and tongue is described under Disorders of Digestive Tract (Chapter VIII.).

a. SORE THROAT is indicative mainly of disease of the *pharynx*, tonsils, and structures around. If the patient complains of "sore throat" turn to § 111, p. 227.

b. HOARSENESS AND OTHER ALTERATIONS OF THE VOICE are  
C.M.

indicative of some affection of the *larynx* (§ 121, p. 237). If NASAL INTONATION OR NASAL DISCHARGE be present turn to § 131, p. 251.

There are also several minor symptoms which arise in conjunction with these, such as a dryness accompanied by tickling



Fig. 50.—NASAL AND BUCCAL CAVITIES, showing the method of LARYNGOSCOPIC EXAMINATION. The three turbinate bones are seen, the anterior end of the inferior turbinate bone having been removed, to show the inner opening of the lachrymal duct. The opening of the Eustachian tube is just behind the posterior end of the inferior turbinate bone. The exact position of laryngoscopic mirror in examination of larynx is shown, namely, over the root of the uvula.

sensations, or an excessive secretion, which leads to “hawking” and “coughing.” Thus it happens that we may be consulted for what the patient believes to be pulmonary disease, when in reality the lungs are perfectly healthy. Dyspnoea and dysphagia may also be produced by local conditions of the throat and larynx. “Globus,” or paroxysmal sensations of a ball in the throat, is a symptom of hysteria.

§ 110. *CLINICAL INVESTIGATION*.—The anatomy and relations of the throat are indicated in Fig. 50; the various parts may be investigated by (a) Direct, and (b) Indirect (*i.e.* Laryngoscopic) examination.

a. For the DIRECT EXAMINATION of the fauces and structures around all that is necessary is a good light and a spatula or spoon to depress the tongue. If direct daylight is not available, as for instance when the patient is in bed, a laryngoscopic mirror can be used (*vide infra*), or a wax vesta, with or without a bright spoon behind it to act as a reflector. The patient should be instructed *not to strain, and to "breathe quietly in and out."* The posterior wall may be seen by directing the patient to say Ha—ah, in which procedure the soft palate is raised. Note should be made of the colour of the mucous membrane, the presence of exudation or ulceration, of granulations or adenoid vegetations in the pharynx, of any mucous patches such as occur in syphilis, or any bulging of the pharyngeal walls. The size and length of the uvula should always be observed, for a long uvula may be the sole cause of chronic cough and numerous otherwise unexplained symptoms.<sup>1</sup>

(b) The INDIRECT or LARYNGOSCOPIC EXAMINATION of the throat will be given below, prior to modifications of the *voice* (p. 237).

§ 111. *CLASSIFICATION, DIAGNOSIS, PROGNOSIS AND TREATMENT*.—**Sore Throat** is a symptom common to nearly all diseases of the throat. Mentioned in order of frequency the diseases which may give rise to it are as follows (*laryngeal affections being excluded for the present—see § 121*)—

*Commoner causes.*

- I. Catarrhal pharyngitis, including two acute and three chronic varieties.
- II. Tonsillitis.
- III. Scarlet Fever.
- IV. Diphtheria.
- V. Syphilis.

*Rarer causes.*

- VI. Other Specific Fevers.
- VII. Cancer, Tubercle, and other new growths.
- VIII. Retro-pharyngeal abscess or Tumour.
- IX. Phlegmonous sore throat.

<sup>1</sup> Whenever a patient complains of cough, coming on, or getting worse, at night or when he lies down, elongated uvula should be suspected. It does not follow that such an uvula may appear too long at the time of inspection. Temporary congestion from various causes, *e.g.*, much talking, produces undue elongation and nocturnal cough. Painting with tannin may reduce it, but the proper treatment is amputation, and it is wonderful what immediate relief is obtained.

§ 112. I. **Acute Catarrhal Pharyngitis** is an inflammation of the mucous membrane of pharynx, and soft palate; and to a certain extent of the tonsils also. It may be so mild as to cause only slight discomfort in swallowing, dryness of the throat, tickling and hawking; and in such mild cases there is only a moderate congestion of the parts. But in severer cases there are constitutional symptoms of some severity, and locally there may be œdema and ulceration. The temperature in such cases varies from  $100^{\circ}$  to  $104^{\circ}$ . The disease rarely lasts more than a few days, ending generally in resolution; although sometimes it passes into a chronic condition.

**Ia. Hospital Sore Throat** is a severe variety of the preceding, attended by considerable ulceration upon the fauces, tonsils and even pharynx. There is the greatest difficulty in swallowing, speaking, and sometimes in breathing. The submaxillary and cervical glands are enlarged, and there is often considerable pyrexia and constitutional disturbance, the prostration being out of all proportion to the local inflammation.

**Ib. Chronic Catarrhal Pharyngitis** presents the same symptoms as the acute variety, in a milder degree and extending over a longer period of time. It is often known as Relaxed or Relapsing Sore Throat, on account of the chronic congestion of the parts and the consequent predisposition to the repeated occurrence of subacute attacks. It forms one variety of clergyman's or school teachers' sore throat.

**Ic. Granular (Follicular) Pharyngitis** is a *chronic* condition, the local symptoms of which resemble the foregoing, with the addition of visible granulations on the pharyngeal walls due to the enlargement of the follicles<sup>1</sup>; hence it is sometimes called follicular pharyngitis. This is a common condition, and a person who is the subject of it, although apparently in good health, is liable to suffer from repeated attacks of sore throat whenever the weather is damp, or his health a little below par. There is excessive mucous secretion which collects in the throat, especially in the morning, and leads to chronic cough and hawking. When

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<sup>1</sup> The word follicle is applied not only to the lacunar glands or crypts in the tonsil, but also to the localised collections of adenoid tissue found in the posterior wall of the pharynx. These latter, when enlarged, form the "granulations" of the granular pharynx.



the disease has lasted some time the throat becomes dry from atrophy of the follicles (Pharyngitis sicca).

**Id. Granular (Adenoid) Pharyngitis** is another form of *chronic* pharyngitis, due to the presence of adenoid hyperplasia and vegetations in the pharynx and naso-pharynx. They are sometimes confined to the naso-pharynx, and by an ordinary inspection of the fauces little mischief excepting congestion can be discovered. The lymphoid granulations may involve a large part of the naso-pharynx, occurring as a large grooved cushion or pedunculated growth, which on examination can be seen and felt behind the soft palate. This condition is said always to start in childhood. The child *breathes with the mouth open*, and thus acquires a characteristic vacancy of expression. Curiously enough the intellect is sometimes below the average. The voice has a dull or nasal twang, and there are snoring and disturbed sleep. The nares are narrowed, and the palate may be high from the negative pressure in the nose.<sup>1</sup> The condition is a pregnant cause of middle ear catarrh and subsequent deafness. Adenoids in the naso-pharynx are usually accompanied by chronic enlargement of the tonsils. The disease often runs in families.

The *causes of pharyngeal catarrh* vary somewhat in the different varieties, although the several causes are largely interchangeable. (1) There is no doubt that in certain persons exposure to cold and damp is immediately followed by an attack of pharyngitis, but how far this acts as an exciting cause, or whether, as in the possible case also of tonsillitis, the condition is set up by a microbe which thrives under certain atmospheric conditions, is not yet determined. (2) Unhygienic surroundings, such as bad drains, the atmosphere of a hospital and the like, may certainly give rise to a very severe ulcerating pharyngitis (*e.g.*, hospital sore throat), and here again the cause may be a microbe. The same condition may arise in private houses in which the drainage is out of order. (3) Bad health in the individual affords undoubtedly a predisposition to the disease, and especially to granular pharyngitis—so much so that the throat in some persons constitutes a veritable barometer of the state of their health. (4) The gouty and

<sup>1</sup> *i.e.*, the diminished air tension in the nose not counterbalancing the normal air tension on the buccal aspect of the hard palate.

rheumatic diatheses are said by many to offer a predisposition to pharyngitis (compare No. 6, below). (5) Various local conditions, such as nasal obstruction or insufficiency, leading to mouth-breathing; and thus chronic rhinitis and *adenoid vegetations* are potent causes of recurring "sore throats." Excessive use or misuse of the voice (clergyman's and school teachers' sore throat), excessive smoking, the constant use of alcohol, spiced or hot foods, or working in a dust-laden atmosphere, often play an important part. The bristle of a tooth-brush or a fish bone impacted in the pharynx is a frequent though unsuspected cause. (6) I have often met with chronic pharyngitis in people who live too well. The excessive secretion and the perpetual hawking which results have in several instances directed the attention of the patient and of his medical adviser to the throat, larynx, or lungs; but the cure of these cases cannot be accomplished until dietetic and other measures are directed to the relief of the portal congestion. (7) Pharyngitis, especially the chronic forms, is often associated with anæmia, and iron is one of the most valuable remedies we have.

*Prognosis.* Pharyngitis is rarely fatal, but it is one of the most frequent and troublesome of the minor ailments which we are called upon to treat. The milder varieties of the acute pharyngitis last only a few days, but the severer forms, such as hospital sore throat, may last many weeks, and be followed by considerable debility. All of the chronic forms have a great *tendency to relapse*.

*Treatment.* The indications are to relieve the local inflammation, to improve the general condition, and to prevent relapse. For the *acute forms*, most of the remedies mentioned under tonsillitis are available. In all subacute and chronic forms, smoking, alcohol, and other causes of local irritation must be avoided. Excessive secretion may be removed by a gargle of bicarbonate of soda. For the "relaxed throat" a gargle consisting of a wine-glassful of water to which a pinch of salt has been added is useful; so also are gargles of alum, pot. chlor., and ammon. chlor. (F. 15—19). Probably carbolic acid, painted on as glycerine (B.P.), or employed as a spray, gargle or lozenge, is the best application. A good spray is that of menthol, gr. x to the oz. of pareoline. Later, astringent paints should be used, *e.g.*, nitrate of silver (20 gr. to the oz.) or equal parts of tr. of iodine and the glycerine of alum.

The most efficient treatment for the granular forms of pharyngitis, where gargles are of little use, is painting with silver nitrate (grs. 40 or 80 to the oz.), or with liq. ferri perchlor., or iodine in glycerine. Trichloroacetic acid is recommended highly. In cases of dry pharyngitis the ammonium chloride inhaler or lozenges are very useful. All these measures, however, may give only temporary relief to the *adenoid* variety, and then scraping away the vegetations is necessary. For a permanent and radical cure the vegetations must be thoroughly removed under general anæsthesia. Nasal obstruction, if present, must also be relieved.

The general health in certain forms, especially the granular varieties, is often of more importance than the local condition, and many a relapsing and granular pharyngitis can be cured by Blaud's pills. Any rheumatic or gouty diathesis should receive attention, and dyspepsia or constipation, especially if associated with portal congestion, should be appropriately treated.

§ 113. II. **Tonsillitis**, or inflammation of the tonsil, is met with clinically in three forms—*a* and *b*, Two varieties of *Acute Tonsillitis*, and *c*, *Chronic Tonsillitis*. *Peritonsillitis* is sometimes described as a variety; it is an inflammation of the connective tissue in the vicinity of the tonsil; it accompanies catarrhal pharyngitis, and is sometimes due to decayed teeth.

The two forms of **Acute Tonsillitis** are as follows :—

**IIa. Acute Parenchymatous Tonsillitis** (Quinsy, Acute suppurative Tonsillitis). The symptoms are pain, swelling, and redness of the tonsils, coming on more or less suddenly with constitutional disturbance, the temperature varying from 101° to 104°. One tonsil is usually more affected than the other, and there is pain, stiffness, and tenderness behind the angle of the jaw. The disease usually subsides in the course of a week; if it last longer than this, suppuration has almost certainly occurred on one or other side. This is evidenced by the increased enlargement, by the swelling spreading along the soft palate, backwards, and downwards into the pharynx. The abscess usually bursts in the course of one or two weeks into the pharynx, but it may occasionally point in other directions.

**IIb. Acute Follicular Tonsillitis** is of a more superficial character. It is attended by the same symptoms as the foregoing, with the exception that abscess rarely occurs; and the surfaces

of the tonsils present numerous yellow points of thick purulent secretion, and perhaps ulceration. There are usually less fever and pain than in IIa. above.

The *diagnosis* of both these forms of tonsillitis from scarlet fever and diphtheria is sometimes a matter of considerable difficulty, but one of great importance ; it is given in the form of a table (p. 234).

*Etiology.* The function of the tonsils is still a matter of some uncertainty, and therefore it is not surprising that the etiology of tonsillitis is largely speculative. 1. Hereditary predisposition seems to play its part, though this usually takes the form of an hereditary gouty or rheumatic diathesis. 2. Unhygienic conditions, and especially bad drainage, have been credited with causing the disease. 3. The tonsils become acutely inflamed in all cases of scarlet fever, in diphtheria, and in so large a proportion of cases of rheumatic fever that the tonsils might be regarded as the portal of entrance of the virus of that disease. The association between tonsillitis and the latter disease has been the matter of much discussion. 4. Cold and damp weather are certainly conditions under which tonsillitis more frequently arises. 5. Traumatism, such as drinking out of a boiling kettle. Fish bones and bristles of a tooth-brush sometimes give rise to one-sided tonsillitis.

**IIc. Chronic Tonsillitis** (chronic enlargement of the tonsils) occurs in two forms. That form seen chiefly in adults after repeated attacks of acute tonsillitis is due to a fibroid degeneration, and is known as Relapsing Tonsillitis, or Chronic Fibroid Tonsillitis. The other and commoner form is that seen in children, which consists of a *parenchymatous* hyperplasia. The condition is almost always associated with adenoids in the naso-pharynx, and consequently there are snoring and mouth-breathing.

*Course and Prognosis.* Acute Tonsillitis is a frequent, sometimes a very troublesome, but never a fatal disease. Sometimes the patient continues at work, but at others he is totally incapacitated. The chief consequence to be feared is constant recurrence, and this leads to chronic enlargement of the tonsils. Chronic Tonsillitis is a troublesome condition because it renders the patient so liable to repeated attacks of acute tonsillitis and coryza. It is a common source of recurrent pharyngitis, leading to otitis media and deafness. Enlarged tonsils met with in children



occasionally disappear during adolescence ; but in some way, only imperfectly explained, the mental and physical development of children who have chronic enlargement of the tonsils is sometimes impeded. Bearing in mind the large quantity of lymphoid tissue which these structures contain, this circumstance is possibly explained by a defective hæmopoietic function. It is, however, doubtful whether the development of the child is hindered unless there be concurrent adenoids, which interfere with the respiratory or oxidative processes of the body.

*Treatment.* The indications are (a) to reduce the local congestion, (b) to reduce the pyrexia, and (c) in chronic tonsillitis to prevent relapse.

(a) Powdered sod. bicarb. applied directly to the tonsils has been credited with aborting the disease. A cocaine spray (4 per cent.) relieves the pain. Cold compresses externally, steam inhalations, warm gargles of pot. chlor., sod. bicarb., salol, and weak alum or carbolic acid (1 in 100) relieve the congestion (F. 15 to 19). In sub-acute or relapsing cases, the tonsil may be painted with pot. iod. gr. 15, iodine gr. 12, ol. menth. pip. ℥ij, glycerine ʒj.

(b) To reduce the pyrexia a brisk saline purge should be given at the outset. Tr. aconiti, ℥i doses, may be given every half hour during the first few hours ; then sod. salicylate as in rheumatism, salol, or liq. ferri perchlor. If the disease does not clear up in a week one may be almost sure an abscess has formed, and should be incised. This is best done with a curved bistoury, round which lint is twisted to within an inch of the point, which should be directed *inwards and backwards* to avoid the internal carotid. Make a small incision, then insert a probe or dressing forceps and push it in all directions.

(c) In chronic tonsillitis the most useful remedies are iron, quinine, cod-liver oil, and other tonics. Salicylic acid, guaiacum and colchicum are used in the Relapsing form. The chronic enlargement may be relieved by painting the throat with glycerine of tannic acid (a most nauseous preparation), or tr. of iodine (ʒj - ʒviij), and other astringents (*vide supra*). . But in most of these cases the question of tonsillotomy arises sooner or later. Parents sometimes raise objections on the score that it may "impair the voice" or "injure the health," but there is no reason to believe this is so.

§ 114. III. In **Scarlet Fever** the tonsil is generally the chief seat of inflammation in the throat. Both have a more or less sudden onset with constitutional symptoms, and thus the diagnosis is often one of considerable difficulty. There are four distinguishing features of scarlet fever, viz. (i.) The diffuse *scarlet* colour of the soft palate and pharynx, with complete immunity of the larynx; (ii.) sudden onset of the illness with high fever; (iii.) on the second day the rash; and (iv.) about the third day the "strawberry" tongue. (See table below, and Chapter XV.)

TABLE XII.

*Tonsillitis.**Scarlet Fever.**Diphtheria.*

## a. LOCAL SIGNS.

Swelling and redness chiefly confined to one or both tonsils. In Follicular T., tonsils covered with sticky mucus, and with numerous, small, separate yellow spots of secretion on one or both, which are easily removable. Nothing on soft palate. Pain severe.

Diffuse *bright* redness of throat generally. The tonsils swollen, and may be covered with mucus, and *sometimes* with multiple yellow points. Nothing on soft palate in ordinary cases.

Ashy-grey patch or patches on tonsils, uvula, and *soft palate* (latter situation is *pathognomonic*); patches *larger* than the follicular secretion in tonsillitis. Patches consist of membrane surrounded by red areolæ; difficult to remove, leaving raw surface. Klebs-Löffler bacillus found in membrane. Sometimes a muco-purulent, acrid *nasal discharge*. Comparative absence of pain.

## b. GENERAL SYMPTOMS.

i. Onset moderately sudden, with moderate fever.

ii. Temperature may be very high, but local symptoms are more troublesome than general symptoms.

i. Onset very sudden, with high fever.

ii. Temperature very high. Local symptoms a subordinate feature.

iii. Rash on second day.

iv. Strawberry tongue about third day.

i. Onset insidious. Early and marked enlargement of cervical glands.

ii. Temperature not so high at first, and may remain low during whole course.

iii. Paralytic sequelæ sometimes.

§ 115. IV. The sore throat of **diphtheria** may be recognised at once if there be an ashy-grey patch of exudation *upon the soft*

*palate*. When this is absent it is chiefly with Follicular Tonsillitis that difficulties arise. In diphtheria the large size and the colour of the patches (which are grey with surrounding red areolæ), the difficulty of removing them, and the raw bleeding surface left, enable us to come to a conclusion. The onset is more insidious, the pyrexia less marked, but the prostration is greater in diphtheria. A muco-purulent, or hæmorrhagic discharge from the nose is very characteristic of diphtheria. The occurrence of albuminuria is given by some as distinctive of diphtheria, but it is very frequently observed in acute tonsillitis also. When other diagnostic features are absent, the presence of *one* large patch on a tonsil, instead of several small patches, is in favour of diphtheria.

§ 116. V. **Syphilitic Sore Throat** is very characteristic. This and the other *secondary* manifestations of syphilis come on about three to six weeks after the appearance of the chancre. 1. The tonsils may be inflamed, but the inflammation is more generalised, and the mucous membrane presents greyish-white semi-translucent irregular patches ("snail-tracks"), on the fauces, tonsils, palate, and other parts of the buccal mucous membrane. Superficial ulceration may also be present, especially on the tonsils, with red punched out edges and yellow-grey secretion. 2. Bilateral symmetry is a very characteristic feature of all these lesions.

*Tertiary syphilitic* ulcers may produce sore throat, their favourite position being the soft and hard palate, the tongue, and the fauces. They are usually preceded by gummatous swellings. 1. The ulcers are deep, with ragged floor, sharply cut edges, and covered with thick yellow-grey secretion. 2. They are progressive, and in course of time will destroy the hard palate or any other parts they invade. 3. They leave characteristic stellate cicatrices which are indisputable evidence of the disorder.

*The less frequent causes of Sore Throat are*—RETRO-PHARYNGEAL ABSCESS, PHLEGMONOUS SORE THROAT, NEOPLASTIC ULCERATIONS, and ACUTE SPECIFIC FEVERS.

§ 117. VI. **Retro-pharyngeal Abscess**, or inflammation of the lymphoid and areolar tissue between the pharynx and the spine, may come on insidiously, or it may be comparatively

sudden. It is known by—1. The rigidity of the head, with difficulty of swallowing, and alteration of the voice; 2. Evidence of swelling in the posterior pharyngeal wall on inspection and palpation, by which it is diagnosed from croup in children.

*Etiology.* Those cases with an *acute* onset are generally either part of a septic inflammation after fevers, or occur in rachitic children under four. Retro-pharyngeal swelling coming on *slowly* is generally due to pus burrowing from some adjacent locality, especially from caries of the vertebræ.

*Prognosis and treatment.* The acute condition is always grave, and requires prompt surgical interference, generally free incision; meanwhile, steam inhalations and warm fomentations relieve the symptoms.

§ 118. VII. PHLEGMONOUS SORE THROAT, *i.e.*, Acute Septic Inflammation of the Pharynx and Larynx.<sup>1</sup> This extremely severe disease may start *inside* the throat, with symptoms of sudden pain accompanied by considerable swelling, leading to severe dyspnœa (stridor), aphonia, and complete dysphagia in a few hours. There is considerable œdema around the fauces, followed by a brawny infiltration of the skin of the neck, spreading from under the jaw to the tongue and larynx. Sometimes the infiltration starts *externally*, and rapidly invades the internal structures. There is much constitutional disturbance, and a temperature of 102°—105°, but unless pus forms rigors and delirium are generally absent. Pus formation is further indicated by a widely and irregularly intermittent pyrexia. Mild cases begin with a stiffness and pain in the tissues around the jaw, and if recovery is to take place the symptoms go no further. But in many cases, and especially in alcoholic and debilitated subjects, the disease rapidly progresses, and death takes place in 12—48 hours, from heart failure, coma, or asphyxia from œdema of the larynx. Suppurative forms are very fatal. Among the recognised complications are pneumonia, pericarditis, pleurisy, and meningitis. The disease is readily recognised by its sudden and severe onset and rapid progress, usually to a fatal issue.

*Etiology.* The condition happily is rare, and the causes consequently obscure. 1. It sometimes arises in association with scarlet fever, erysipelas, and small-pox (in former times being a common cause of death in this disease), or other acute specific fevers. 2. It may arise in people apparently in good health, and has then been attributed to the entrance of a specific microbe by the tonsils, or through the socket of an extracted tooth.

*Treatment.* The indications are to control the inflammation, and to keep up the strength of the heart. An ice-bag should be applied locally. Quinine (iv or v gr.) should be given every four hours. Iron and digitalis are recommended. Free and early incisions should be made at once into the œdematous tissues; and the practitioner should always be

<sup>1</sup> The disease has been variously described by the following names:—Acute Inflammatory Œdema, Erysipelas of the throat, Phlegmonous Cellulitis, Acute Infectious Phlegmon (a term applied by Senator when the inflammation was confined to the wall of the pharynx), Angina Ludovici (when the inflammation is chiefly external—in the neck).



at hand to perform tracheotomy, if the dyspnœa be increasing. Stimulants must be liberally administered. It might be worth while to try the efficacy of antistreptococcus serum.

§ 119. VIII. CARCINOMA frequently and SARCOMA occasionally involve the pharynx either primarily or secondarily. Their diagnostic features are the same as those mentioned under *The Tongue* (§ 149).

TUBERCULOUS ULCERS of the pharynx are rare as primary lesions. 1. They resemble syphilitic ulcers, but there is pallor of the mucous membrane, and a characteristic "worm-eaten" appearance of the pharyngeal wall. 2. *Their course is not nearly so rapidly progressive.* 3. It may be possible to obtain the tubercle bacillus from the scrapings; and 4. there are usually other manifestations of tubercle, especially in the lungs. For treatment see Tuberculosis of the Larynx (p. 243).

§ 120. IX. ACUTE SPECIFIC FEVERS other than those mentioned above, such as typhoid, give rise to inflammation and ulceration of the throat. In variola, for example, the pustules often form upon the palate, fauces and buccal mucous membrane, leaving superficial circular ulcers. An examination of the throat is often useful as an aid to the diagnosis between measles, scarlet fever, and small-pox. The first-named always affects the *larynx*, rarely the pharynx: scarlet fever always affects the *pharynx*, and very rarely the larynx; whereas small-pox affects them *both about equally*. Patches of lichen planus may be found on the palate when the disease exists on the skin; and the eruption of measles may be found in that situation a day or two before it appears on the skin.

### The Larynx.

§ 121. SYMPTOMS and CLINICAL INVESTIGATION.—It will be remembered that the two cardinal SYMPTOMS of diseases of the throat (used in its widest sense) were (a) Sore Throat, and (b) Alterations of the Voice. Both of these may be present in disorders of the larynx, but it is the latter especially which indicates derangements of the organ of voice. Diseases of the larynx are also sometimes indicated by Cough, Hawking, Dysphagia, Dyspnœa, and actual Pain in the organ. But in some cases all of these may be absent; there may indeed be pronounced disease of the larynx (*e.g.*, paralysis or papilloma) without any *subjective* symptoms.<sup>1</sup>

The CLINICAL INVESTIGATION of the larynx (laryngoscopy) is a procedure of considerable technical nicety, and requires some practice. The necessary appliances are a good steady light, a *reflecting mirror* mounted on a band or a spectacle frame for the operator's forehead, and a small circular *throat-mirror* mounted on

<sup>1</sup> Not long ago I met with the case of a well-known operatic singer who had a small papilloma just beneath one vocal cord; her voice was in perfect order, and she could reach the highest notes with perfect ease. The only defect was a hardly perceptible weakness in the middle register.

a handle at an angle of  $135^{\circ}$ . The light should be placed on a level with, and a little behind, the patient's left ear. The operator takes his seat directly opposite; and it is advisable that his seat should be a little higher than that of the patient. Having directed the patient to open his mouth and "breathe quietly in and out," the first step is to adjust the *reflecting mirror* in order to thoroughly illuminate the back of the pharynx. The focal length of the head-mirror is generally 10 to 20 inches, and this should represent the distance of the mirror from the patient's pharynx. Having warmed the throat-mirror over the lamp, the next step is to pull the patient's tongue with the left hand gently out of the mouth with the aid of the corner of a towel or a piece of linen rag. Take the corner of the towel in the right hand, lay it on the patient's tongue, then grasp the tongue and towel firmly between the left thumb and finger. Take care not to hurt the under surface of the tongue against the teeth of the lower jaw. Then, test the warmth of the throat-mirror against your cheek or the back of your hand, and, having pushed the patient's head a little backwards by pressing your right thumb against the upper teeth, introduce the mirror with the right hand, *taking care to avoid touching the top of the tongue* in so doing. Push the mirror obliquely upwards against the soft palate just over its junction with the uvula (Fig. 50, p. 226). This should give you a good view of the vocal cords by slightly lowering or raising the handle. In children and persons with very sensitive throats it is sometimes advisable to anæsthetise the pharynx before laryngoscopy, either by a spray of, or painting with, a 4 or 5 per cent. solution of cocaine, or by the administration of a few doses of bromide during the preceding twenty-four hours.

In normal conditions the *epiglottis*, which is in reality anterior, appears at the *upper part of the mirror*. The *vocal cords*, which are of a pearly white colour, are close together at their upper or epiglottic ends; and at their lower (really posterior) ends are widely divergent during quiet respiration. At their lower ends they appear to terminate in two prominent knobs seen at the lower edge of the mirror, which mark the position of the *arytenoid cartilages* (Figs. 51 and 51a). The *ary-epiglottic folds* stretch on each side from the arytenoids to the sides of the epiglottis. In

these folds, just external to the arytenoid on each side, may be seen a small prominence, the *cartilage of Wrisberg*. To the outer side of the cords lie the ventricular bands or false cords of mucous membrane. With a little practice, and under favourable circumstances, the bifurcation of the trachea may be seen.

In LARYNGOSCOPY there are FOUR MATTERS to be investigated :—

(a) The presence of *congestion* or *pallor* of the vocal cords and the parts around. Congestion of the vocal cords is an evidence of LARYNGITIS ; sometimes of ulceration or new growth.

(b) The presence of any *ulceration*. Ulceration occurring in a person below middle age is very often due either to SYPHILIS or TUBERCLE ; in a person beyond middle life it is not infrequently MALIGNANT.

(c) The presence of a *nodule* or *new growth*. A nodule or new growth turns out most frequently to be a PAPILLOMA or wart of a benign character.

(d) Whether there is any *paralysis* or *spasm* of the vocal cords, which is evidenced by the size, shape, and *mobility* of the aperture.

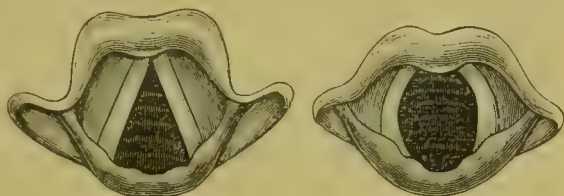


Fig. 51.—Quiet Inspiration. Fig. 51a.—Forced Inspiration.

We shall deal with the disorders of the larynx in this order.

§ 122. CLASSIFICATION.—There may, as just mentioned, be no *subjective symptoms* even in presence of pronounced disease of the larynx, and therefore it will be well to adopt as a basis of classification the *physical signs* discovered by laryngoscopy. However, when any symptoms are present there is always some ALTERATION OF THE VOICE ;<sup>1</sup> and the principal diseases giving rise to such alterations (*i.e.*, the **causes of alterations of the voice**) may be grouped as follows :—

### I. LARYNGITIS—

- (a) *Acute Laryngitis*, including also—  
Edema Glottidis, and  
Foreign bodies in the Larynx or Trachea.
- (b) *Chronic Laryngitis*, including also—  
Perichondritis, and  
Infantile Stridor.

### II. ULCERATIONS of the Larynx—

- (a) Tubercular Ulceration,
- (b) Syphilitic Ulceration,
- (c) Lupus of the Larynx, \*
- (d) Malignant Ulceration.

<sup>1</sup> Excepting, perhaps, bilateral abductor paralysis, in which there may be dyspnoea and stridor without alteration of the voice.

## III. NODULES and NEW GROWTHS—

- (a) Benign (warts and polypi),
- (b) Malignant Neoplasms and Leprosy.

## IV. PARALYSIS of the Vocal Cords—

- Bilateral Abductor Paralysis,
- Unilateral Abductor Paralysis,
- Total (Ab- and Adductor) Bilateral Paralysis,
- Total (Ab- and Adductor) Unilateral Paralysis.

## V. SPASM of the Vocal Cords—

Laryngismus Stridulus or Croup.

## VI. Diseases of the PHARYNX (§ 111); VII. Diseases of the NOSE (§ 132); VIII. Some severe PULMONARY affections; and

IX. Certain NEUROSES also cause alterations in the voice.

I. *The patient complains of huskiness or loss of voice, a comparatively dry cough, soreness on swallowing, and there are local signs of congestion of the vocal cords. The disease is LARYNGITIS, of which two varieties are met with, ACUTE and CHRONIC.*

§ 123. **Acute laryngitis** comes on somewhat suddenly and usually runs its course in a week. It is not usually a serious affection, but in children it may be alarming. In children a slight laryngitis coming on suddenly is a frequent cause of what mothers describe as “croup.” Owing to the dryness of the cords, the child wakes up suddenly at night with loud inspiratory stridor followed by an attack of coughing. This symptom is technically known as *laryngitis stridulosa*, and is not to be confused with laryngismus stridulus (see “Spasm of the Cords”). It is differentiated from membranous croup (laryngeal diphtheria) by the perfect general health of the child in simple laryngitis.

*Etiology.* The chief cause of acute laryngitis is exposure to cold—especially when combined with overuse and misuse of the voice (*e.g.*, actors, music-hall artistes, etc.). Diphtheria or measles may start in the larynx. Persons who suffer from chronic laryngitis (*q.v.*), or nasal obstruction, are predisposed to attacks. A foreign body in the larynx or trachea is a cause of irritation which may produce symptoms resembling laryngitis.

*Prognosis.* The affection is troublesome and apt to recur. When occurring during the course of the specific fevers the prognosis is less favourable, because Edema Glottidis may supervene.

*Treatment.* All use of the voice must be forbidden. The patient must be kept in a warm moist atmosphere, and should



use warm inhalations (such as tr. benzoin co. ʒ j to the pint of boiling water, and see also F. 110). Warm compresses or fomentations should be applied externally, and warm mucilaginous and alkaline drinks should be freely taken. According to some authors, a strong solution of silver nitrate (20 grs. to 3 i.) applied locally at the outset may cut the disease short. For laryngitis stridulosa, hot sponges should be applied to the throat, and vin. ipecac. in teaspoonful doses, with warm water, should be given every ten minutes or so until emesis ensues.

§ 123a. **Œdema Glottidis**, or œdematous laryngitis, consists of an œdematous swelling affecting the epiglottis and submucous tissue of the larynx, but the vocal cords are not involved. The onset is usually sudden, and attended by considerable dyspnœa, dysphagia, and inspiratory stridor. The diagnosis is usually simple on account of the swelling which can be seen and felt on palpation at the back of the tongue. If this be absent some difficulty may be experienced; but the sudden onset of laryngeal dyspnœa should bring the disease to our minds. It may arise either as a primary or as a secondary affection. As a primary disease it may come on as part of an acute septic inflammation of the throat, or it may be part of an œdematous angio-neurosis of urticarial origin (see "*Acute Œdema of the Tongue*"). It may occur as a *secondary* condition in association with (1) one of the various causes of acute or chronic laryngitis; (2) a general anasarca; (3) injury of the glottis by boiling or caustic liquids, etc. Its rapid onset is the chief source of danger, but if the patient does not shortly succumb to asphyxia, recovery generally takes place in a few days.

The *treatment* consists in the administration of emetics, and ice internally and externally. In severe cases, if a 20 per cent. cocaine spray fail, scarification of the epiglottis must be resorted to; and if this be unsuccessful, tracheotomy must be performed without delay.

§ 123b. **The swallowing of a foreign body**, and its passage into the larynx or trachea, has always to be borne in mind in children suffering, apparently, from acute laryngitis; for the history is often wanting. *Paroxysms of dyspnœa* or of *coughing* in a child without obvious cause, should make us suspect it. Unless it has passed into the bronchus (usually the right), a foreign body may be seen by laryngoscopic examination. On the other hand, when a foreign body passes into the bronchus, it may cause so little cough or disturbance at the time that the patient may imagine he has swallowed it, or he may be unmindful of the incident. Obscure cases of unilateral bronchiectasis are probably due to such causes.

§ 124. **Chronic laryngitis** is a more troublesome affection on account of the perpetual hoarseness and liability to acute attacks. Its causes are (1) repeated acute attacks; (2) excessive speaking, singing, teaching, and overuse of the voice (actors, clergymen, school teachers, etc.); it also affects masons, fustian-cutters, and others exposed to dusty atmospheres; (3) nasal obstruction and

mouth-breathing ; (4) tubercle, syphilis, and new growths, the evidences of which should always be sought in cases of intractable laryngitis. These usually go on to ulceration, under which they will be described. (5) Spread of inflammation from adjacent parts. Many cases of chronic laryngitis depend upon a granular condition of the pharynx. (6) Rheumatic and gouty diatheses predispose.

*Treatment.* The indications are to obviate the cause and to relieve the local congestion. The removal of the cause is most important, and often most difficult to accomplish, for a large number of the patients are singers, teachers, and others whose living depends upon the daily excessive use of the voice. The avoidance of tobacco and alcohol will aid, and residence in a dry climate will often accomplish a speedy cure. Much may be done to prevent or relieve the condition by proper voice-production and respiration.<sup>1</sup> Locally, paintings with strong astringent remedies, such as zinc chlor. (gr. 30 to the oz.) or silver nitrate (gr. 20 to 60 to the oz.), are useful. These strong applications should not be made more than twice a week ; weaker solutions can be applied more frequently. The patient himself may use sprays of alum (gr. 5) or zinc sulphate (gr. 2 to the oz.) for five minutes twice daily, or inhalations of turpentine, creasote, iodine, menthol, &c., for fifteen minutes three times a day.

§ 124a. **Perichondritis**, *i.e.*, inflammation of the perichondrium of the laryngeal cartilages. Opinions differ as to the frequency of this affection. If considerable, it may lead to necrosis of the cartilages and abscess of the larynx. The differential features, besides loss of voice or hoarseness, are dull aching pain and acute tenderness. This may be accompanied by swelling in the neck. As regards its *etiology*, apart from traumatism, it is rarely a primary malady. It more often occurs secondary to syphilitic or tubercular laryngitis. Syphilis is its commonest cause. It also follows enteric fever.

*Prognosis and treatment.* It is a serious affection, for even in the mild forms the voice is rarely restored. Great stenosis of the larynx may result. If there be much swelling the dyspnoea is very marked ; and the patient may die from pneumonia or gangrene of the lungs, or, in the suppurating forms, from pyæmia. Abscess and fistula may follow.

§ 124b. **Chronic infantile stridor** is a term applied to more or less continuous inspiratory dyspnoea, accompanied by a croaking sound, occurring for the most part in infants. It may be more or less constant up to the age of two, or occur only at intervals during that period of life. It is believed to be caused by a folding of the epiglottis, possibly due to

<sup>1</sup> This affection is extremely common among our school board teachers, owing chiefly to faulty voice-production, and they ought to be specially trained to obviate this defect. The Rev. J. P. Sandlands, M.A., Brigstock, Thrapston, devotes himself to the cure of such cases.

some malformation. It is usually attended by a certain amount of laryngitis, and hoarseness.

II. **Ulcerations** of the larynx are met with chiefly in two affections, syphilis and tubercle; and, in persons past middle life, malignant disease may be a cause. The simple erosions present in catarrhal laryngitis hardly amount to ulceration. Ulceration is also found with Lupus and Leprosy, usually when cutaneous lesions are present.

§ 125. **Chronic Tuberculous Laryngitis** should always be suspected when delicate patients complain of constant hoarseness. This form of laryngitis is recognised by (1) the general pallor of the mucous membrane, accompanied by a thickening or swelling most marked over the arytenoids or the aryteno-epiglottic folds; (2) the occurrence of irregular, slowly growing ulcers, usually bilateral; and (3) the history or presence of pulmonary tuberculosis.

The *prognosis* is always grave, and until recently recovery when the larynx was involved in tuberculosis was practically unknown. The course of the affection depends more upon the condition of the lungs (§ 98) than that of the larynx.

The *treatment* at first is largely constitutional, *e.g.*, creasote in doses of  $\text{m i}$  to  $\text{v}$  is recommended (*vide* Phthisis). Locally menthol, one part to five of olive oil, used as a paint; or an insufflation of menthol, gr. 8, with iodoform and boracic acid, of each  $\text{3 i}$ , is valuable. When ulceration has occurred, after being swabbed with cocaine and curetted, the parts should be thoroughly brushed with lactic acid, 10 to 60 per cent. This is a very favourite application. For the pain, which may be severe enough to cause dysphagia, morphia gr.  $\frac{1}{4}$ , with starch gr.  $\frac{1}{2}$ , may be blown into the larynx; or a 10 per cent. cocaine spray. Rest and a warm, dry climate are indicated, and sanatorium treatment (§ 98).

§ 126. **Chronic Syphilitic Laryngitis.** The laryngitis accompanying secondary syphilis may resemble simple catarrh, with the addition of whitish patches (§ 116). But that which occurs in the later stages nearly always takes the form of ulceration. The intensity of hyperæmia, the irritability, and the profuseness of the purulent discharge are features of syphilitic ulceration. It is distinguished from the tubercular ulceration by

(1) the bright red coloration of the mucous membrane ; (2) the presence of a deep, *rapidly growing ulcer*, with bright yellow surface, regular edges, often undermined, sometimes unilateral. If the ulcers invade the upper surface of the epiglottis this is said to be pathognomonic of syphilis ; (3) the presence of a syphilitic history.

*Prognosis and treatment.* This form of laryngitis is twice as rapid as, and far more destructive than, the preceding, and is sometimes liable to involve the cartilages (*vide* Perichondritis). Even when arrested considerable stenosis may result. The usual constitutional treatment must be carried out, full doses (60 to 100 grains) of pot. iodide being given. Local applications of iodoform or a spray of perchloride of mercury, 1 in 1,000, are employed.

(c) **Lupus** of the larynx occurs usually after the throat has been primarily attacked by the disease, and is known by—1. The distortion of the parts about the pharynx, with nodules. The disease progresses very slowly. Ulcers may be seen healing at one place and spreading at another. 2. There is usually but little pain. 3. There is no secretion, such as exists in chronic simple pharyngitis. 4. There is often present cutaneous lupus, which aids the diagnosis considerably.

(d) **Malignant disease**, and (in other countries) **Leprosy**, give rise to ulceration of the larynx. (See below.)

III. **Nodules and new growths.** *Flat localised thickenings of the mucous membrane are spoken of as warts, nodes, or nodules. When they are pedunculated they are spoken of as polypi. In either case they begin most frequently as a unilateral thickening on or near one of the vocal cords. In the early stage they are extremely difficult to distinguish from syphilis or tubercle, and sometimes this can be accomplished only by the history. With one important exception (singer's node), nodules are UNILATERAL, and this feature of asymmetry distinguishes them from the thickening which may result from chronic laryngitis. The practical point of prime importance is the distinction of Benign from Malignant growths, although this again is often a task of considerable difficulty.*

§ 127. **Benign new growths** begin most frequently as **warts**, nodules, or thickenings, the surface of which is smooth, although congested. They may give rise to no symptoms for a considerable time, unless they happen to be on the free edge of the cord. Perhaps the commonest of these growths is what is known as a **singer's node**. This lesion very often affects the under surface of the vocal cord, and hence may be overlooked for a long time. It is



distinguished from other nodules by its frequent involvement of both sides symmetrically. A projection on one cord at the junction of the *anterior with the middle third* is probably a Singer's Wart; one situated at the junction of the *posterior with the middle third* is probably pachydermia laryngis: in the latter case there is a nipple on one cord which fits into a crater on the other. **Pachydermia Laryngis** is a localised ch. laryngitis (§ 124), usually most marked over the vocal processes. *Benign* nodules as a class are differentiated from *malignant* by the absence of pain and the paucity of symptoms of any kind. A pedunculated benign growth, **polypus**, of the larynx has the same clinical features, but is accompanied by very characteristic attacks of paroxysmal dyspnœa.

§ 128. **Malignant growths** start as thickenings of the mucous membrane, which may be like those of the preceding class, or they may be greyish white, or they may have a ragged surface. In the course of a few months they invariably go on to erosion and ulceration. Malignant are differentiated from the benign growths by (1) the age of the patient and the family history; (2) the early onset of hoarseness and sometimes pain which may have preceded any other symptom for many months; and (3) their rapid and progressive nature, giving rise in the course of a few months to foul breath and hæmorrhage. A diffuse unilateral congestion or *unilateral paralysis* in a person over middle age is always suggestive of malignant disease.

*Prognosis and treatment.* *Benign* growths often cause but little inconvenience. They are generally removable, without ulterior damage, by snares or cutting forceps. The *malignant* growths constitute one of the most rapid and fatal forms of the disease. Early extirpation of the larynx offers the best prospect, and in the case of Montague Williams (whose larynx is now in St. Thomas's Hospital Museum) afforded immunity for two or three years.

IV. **Paralysis of the vocal cords** can be detected only by carefully inspecting both the POSITION and the MOBILITY of the cords during (i.) rest, (ii.) phonation, and (iii.) deep inspiration.

§ 129. **Paralysis of the Vocal Cords.** The chief actions of the larynx are (i.) Abduction (glottis-opening), which is performed by the

posterior crico-arytenoids, and (ii.) Adduction (glottis-closing), which is performed by the lateral crico-arytenoids and the arytenoideus muscle. The cords are rendered tense by the crico-thyroids (external tensors), and are relaxed and shortened by the thyro-arytenoids (internal tensors, *i.e.*, the muscle which lies in the vocal cord). The larynx is supplied by two nerves, the superior laryngeal and the recurrent laryngeal branches of the vagus. The former supplies the crico-thyroid or tensor muscle and the mucous membrane of the larynx, while the recurrent laryngeal supplies all the other muscles. In progressive lesions of the recurrent nerve the abductors are paralysed first, and later on the adductors.

*The signs of laryngeal paralysis.* It is very rarely that a single muscle is paralysed; the paralysis nearly always affects a physiological group of muscles, *i.e.*, the glottis openers (abductor paralysis), or glottis closers (adductor paralysis), on one or both sides. Paralysis is often accompanied by more or less catarrh, which modifies the appearance somewhat, but the evidences of laryngeal paralysis depend upon the position and mobility of the cords during phonation and respiration. The symptoms are given in Table XIV. (p. 247).

Normally during rest the cords are midway between opening and closing (Fig. 53); during phonation they are approximated so that practically no space is left between them (Fig. 54). During deep inspiration they are widely opened (Fig. 51A).

When the cords are normal during phonation, but do not move out on inspiration, there is bilateral paralysis of the glottis openers (bilateral *abductor paralysis* (Fig. 55)). If both cords move during phonation, but one of them fails to move out fully during inspiration, there is *unilateral abductor paralysis* (Fig. 56).

TABLE XIII.

NAME OF MUSCLE.	NERVE SUPPLY.	ACTION.	
		Phonation.	Respiration.
<i>Crico-thyroid</i> <i>or external tensor.</i>	Superior laryngeal.	Tense and elongate the vocal cords.	_____
<i>Thyroid-arytenoid proprius</i> <sup>1</sup> <i>internal tensor in cord itself.</i>	Recurrent laryngeal.	Adjusts edges of the cords.	_____
<i>Post. crico-arytenoid.</i>	Recurrent laryngeal.	_____	Abduct, <i>i.e.</i> , open glottis.
<i>Lateral crico-arytenoid.</i>	Recurrent laryngeal.	_____	Adduct, <i>i.e.</i> , close glottis.
<i>Arytenoideus.</i>	Superior laryngeal and recurrent laryngeal.	Close the glottis (posterior third chiefly).	_____

<sup>1</sup> Lateral thyro-arytenoid is the lateral part of this muscle.

When the cords neither move to the middle line with attempted phonation, nor move as far outwards as normal during deep inspirations, but remain midway between the two in the cadaveric position (Fig. 53), there is *total bilateral paralysis of adductors and abductors* (Fig. 57).

If during phonation and inspiration one cord remains immobile, there is *total unilateral paralysis*.

If there is aphonia, and on laryngoscopic examination the cords do not meet properly during attempted phonation, although they move outwards with inspiration, there is *bilateral adductor paralysis* (Figs. 58 and 59).<sup>1</sup>

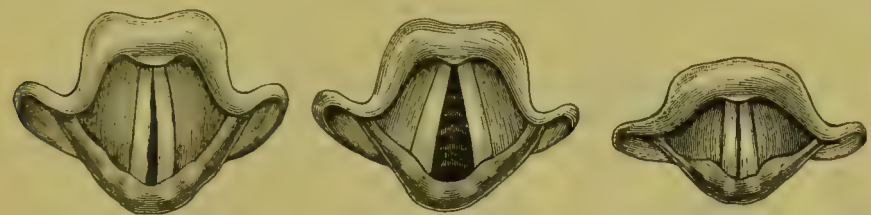


FIG. 52.—MODERATE ABDUCTION. The appearance seen during REST.      FIG. 53. — CADAVERIC POSITION of cords.      FIG. 54. — Typical position during PHONATION of high notes.

TABLE XIV., LARYNGEAL PARALYSES.  
(From Gower's, slightly modified.)

Lesion.	Symptoms.	Signs.
Bilateral abductor (opener) paralysis	Voice little changed; cough normal; inspiration difficult and long, and attended with loud stridor.	Both cords near together; not separated during inspiration, but even drawn nearer together.
Unilateral abductor (opener) paralysis.	Symptoms inconclusive; little affection of voice or cough. Brassy cough sometimes.	One cord near the middle line not moving during inspiration, the other normal.
Total bilateral paralysis.	No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless (i.e., the cadaveric position).
Total unilateral paralysis.	Voice low-pitched and hoarse; no cough; stridor absent or slight whilst breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.
Bilateral adductor (closer) paralysis. <sup>2</sup>	No voice; normal cough; no stridor or dyspnoea.	Cords normal in position, and moving normally during respiration, but not brought together on an attempt at phonation.

The *etiology* of laryngeal paralyses differs considerably in the various forms. They may arise from ORGANIC or FUNCTIONAL conditions; but each is so characteristic that it can be readily identified. Thus *hysterical paralysis* is always double, and very nearly always due to adductor paralysis. Abductor paralysis is generally—and if unilateral is always—organic in origin. If the left vocal cord cannot be abducted it is almost certainly due to pressure on the left recurrent laryngeal, and this in nine cases out of ten is due to *aneurysm of the aorta*.

<sup>1</sup> Further particulars of the actions of the various muscles may be found in a study of laryngeal paralyses since the introduction of the laryngoscope, by Sir Felix Semon, Brain, 1892, vol. xv., p. 471.  
<sup>2</sup> *Unilateral adductor paralysis* is practically unknown, though tubercular or rheumatic ankylosis of the crico-arytenoid joint may cause immobility of one cord.

(a) **BILATERAL ABDUCTOR PARALYSIS** (Fig. 55) may be due to—

- i. The earlier stages of *pressure* upon both recurrent laryngeal nerves, as by mediastinal tumour, or pericardial effusion (§ 57).
- ii. *Peripheral* neuritis from toxins, such as diphtheria, alcoholism, influenza; certain drugs, *e.g.*, lead, arsenic; or simple catarrh.
- iii. *Central* causes, as in lesions affecting the medulla or base of the brain, bulbar paralysis, cerebral tumours or syphilis, hæmorrhage into the bulb, tabes dorsalis, disseminated sclerosis, meningeal conditions, etc.

(b) **UNILATERAL ABDUCTOR PARALYSIS** (Fig. 56) is due to the same causes acting on one side only. Thus, if on the *left side*, it is due in nine cases out of ten to *aneurysm of the aorta*, although no other signs of that condition may be present. Malignant tumour of the œsophagus may also affect the left recurrent laryngeal. Thickened right pleura may be the cause of a paralysed right recurrent laryngeal. Pressure upon the *vagus* in the neck as by an enlarged thyroid, or cervical glands may affect one or both sides.



FIG. 55.—**BILATERAL PARALYSIS OF THE GLOTTIS-OPENERS (BILATERAL ABDUCTOR PARALYSIS).** The patient is able to oppose the cords during phonation, but the cords do not move outwards during deep inspiration (as in Figs. 51 and 51a).

The same appearance as the above is sometimes produced by acute laryngeal catarrh, but the cords would be pink instead of white.

(c) **TOTAL (AB- AND ADDUCTOR) BILATERAL PARALYSIS** (Fig. 57) is practically always of organic origin, but it may (rarely) be due to catarrh or hysteria. It may arise from any of the causes mentioned under bilateral abductor paralysis, but is most frequently of *central* origin. It occurs later in the disease than abductor paralysis, the abductor fibres in the nerve being the first to be affected.

(d) **TOTAL (AB- AND ADDUCTOR) UNILATERAL PARALYSIS** (Fig. 56, during phonation also) is due to the same causes as mentioned under unilateral abductor paralysis, *i.e.*, usually, pressure upon the recurrent

laryngeal. This condition, however, occurs at a later stage in the case, unilateral abductor paralysis being a feature of the earlier stage. Total paralysis is sometimes called "recurrent paralysis," because it is due to paralysis of the recurrent laryngeal.

(e) **BILATERAL ADDUCTOR PARALYSIS** (Figs. 58 and 59) is always *functional* (*viz.*, unconnected with *gross lesions*):—(1) hysterical; (2) simple catarrh, or overuse of the voice; (3) general weakness, as in anæmia. But the first of these is by far the most common.

**Prognosis.** Laryngeal paralysis is generally only a minor element in the case. When occurring alone, however, the prognosis in adductor paralysis is good, because it is always of functional origin, or in the paralysis arising from syphilis, if treated early. In all forms, however, the prognosis depends upon whether the cause is removable or not.

**Treatment.** Hysterical paralysis should be treated on lines laid down elsewhere. Strong faradisation or static electricity to the larynx is indicated in the hysterical variety, the patient being instructed to call out loudly. In organic paralysis the prognosis depends upon the cause. Pot. iod. should receive a fair trial. Strychnine and electricity are



useful. In organic cases, if dyspnœa be severe, tracheotomy must be performed.

ILLUSTRATIONS OF LARYNGEAL PARALYSES.—It should be remembered, in studying these illustrations, that to test the motor power of the vocal cords it is necessary to make the patient **INSPIRE** deeply to **OPEN** the cords, then to **PHONATE** so as to **CLOSE** the cords; and a given position of the cords conveys no information unless it is first known which of these acts the patient is performing.

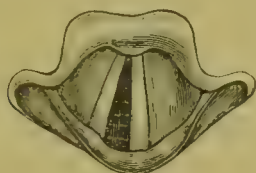


FIG. 56.—LEFT ABDUCTOR, or glottis opener, paralysis. DURING INSPIRATION the *left cord* remains fixed, instead of moving outward like the right cord does. This occurs in early paralysis of the recurrent laryngeal nerve of **ORGANIC ORIGIN** (e.g., aneurysm).



FIG. 57.—TOTAL BILATERAL paralysis. DURING INSPIRATION and DURING PHONATION *both cords* are immobile, and remain in what is practically the cadaveric position. Nearly always of **ORGANIC ORIGIN** and frequently central.

PATIENT'S RIGHT.

PATIENT'S LEFT.



FIGS. 58 & 59.—PARTIAL BILATERAL ADDUCTOR, or glottis closer, paralysis. It is the condition commonly met with in hysterical or FUNCTIONAL aphonia. DURING PHONATION the cords close anteriorly and posteriorly, but leave an elliptical space between them. The glottis is closed by two muscles, the crico-thyroid in front and the arytenoides behind. If the CRICO-THYROID is mainly affected, the condition depicted in Fig. 58 is seen; and it is met with in functional aphonia and exhaustion. The ARYTENOIDEUS closes the posterior angle, and when this is paralysed the posterior angle remains open (Fig. 59). Both of these forms are met with in acute and chronic laryngitis, and are generally independent of any actual nerve lesion, excepting perhaps peripheral neuritis and some rare cases due to a local lesion affecting the recurrent laryngeal nerve of both sides.

In laryngeal paralysis it is very important to decide whether a functional or organic cause is in operation, and the following hints should be remembered:—

- (1) Glottis closer (adductor) paralysis is generally functional; glottis Opener (abductor) paralysis generally **Organic**.
- (2) Bilateral paralysis is generally functional; One-sided paralysis is generally **Organic**.
- (3) Left Abductor (glottis opener) paralysis suggests **Aneurysm**.

V. SPASM OF THE LARYNGEAL MUSCLES, *and consequent* INSPIRATORY DYSPNŒA, is not a very common occurrence, excepting in the form of *Laryngismus Stridulus*, a disease almost confined to childhood. It may arise when a foreign body passes into the larynx, and may occasionally occur in adults who are the subjects of acute laryngitis. *Inspiratory dyspnœa* may also arise in *Bilateral Abductor Paralysis*.

§ 130. **Laryngismus stridulus** or **nervous croup**<sup>1</sup> is a form of paroxysmal inspiratory dyspnœa. It consists of a sudden spasmodic closure of the glottis, followed by a long, noisy inspiration which produces a crowing sound; due to spasm of the adductors. It is a nervous affection, and appears to be due to some irritation of the vagus or of its recurrent laryngeal branch. The whole attack lasts from a few seconds to a minute or two. The child may become cyanosed, or the spasms may spread to other muscles, and give rise to general convulsions. Occasionally it terminates fatally. The attacks come on either during sleep, or in the waking state. They are very apt to recur, and the severity of the attacks may increase at each recurrence. On the other hand, if the attacks are slight, they may gradually disappear as the child grows older. In the intervals the child is free from cough or hoarseness, and the larynx appears healthy.

The *etiology* is obscure. It is practically confined to children of from four months to two years old, and nine-tenths of these are rachitic; that is to say, children in whom infantile convulsions and tetany are also apt to arise. It is twice as common in boys. It is more frequent in the spring time, and it is often hereditary. In older subjects laryngeal spasm and inspiratory dyspnœa occur sometimes in *tabes dorsalis*, when it forms the laryngeal crisis of that disease. Its rarer causes are epilepsy, hysteria, tetany, chorea, reflex irritation of the vagus or its recurrent laryngeal branch from mediastinal growths, a growth or foreign body in the larynx, or too long a uvula.

The *diagnosis* is not difficult, though it is well to bear in mind the possibility of a foreign body in the throat, larynx or trachea. There are, however, three pathological conditions to which the term "croup" is loosely applied, and which consist essentially of a PAROXYSMAL INSPIRATORY DYSPNŒA.

1 Synonyms—*Spasmus glottidis*; spasmodic croup; child-crowing; spasm of the larynx.

1. *Nervous croup* or *laryngismus stridulus* is the non-inflammatory nervous affection described above. This is recognised by the absence of cough, hoarseness, and other symptoms referable to the larynx in the intervals between the attacks. There is often a history of similar attacks.

2. *Catarrhal croup*, or spasmodic laryngitis, consists of attacks of dyspnœa, coming on for the most part at night, in children suffering from a little cough and hoarseness during the day, and lasting for an hour or so. This is due to the collection of thick secretion, or to the sticking together of the edges of the glottis from slight laryngeal catarrh. It should not be confounded, as it often is, with nervous croup.

3. *Membranous croup*, or laryngeal diphtheria. This is true diphtheria, and is attended by the constitutional and other symptoms of that disease (Chap. XV.). However, some (*e.g.*, Whitla and others) maintain that a non-diphtheritic membranous croup may occur. This is doubtful, although a severe injury (*e.g.*, drinking out of a boiling kettle) may certainly result in a membranous or "diphtheritic" inflammation of the mucous membrane.

*Treatment of Nervous Croup.* (a) *For the attacks*—Cold water may be dashed in the face, or the patient plunged into a hot bath, or alternately hot and cold; or cold water douches applied. The inhalation of chloroform relieves it promptly. Artificial respiration is often of great service, and it may restore, even after apparent death. In the rare cases in which the spasm is prolonged and continuous, tracheotomy may be necessary.

(b) *For the intervals*—The patient should be kept very quiet, and irritation of the surface or the application of any stimuli conducive to an attack should be avoided. Reflex causes of irritation should be sought in the gums (*e.g.*, teething), in alimentary canal (*e.g.*, worms or gastric disorder), in the lungs and elsewhere (*vide* causes). The general treatment of rickets should be adopted, and it is worth bearing in mind that children taken into the country very often cease to have these attacks. Sponging with cold water twice or three times a day is of value; and as to medicines, bromides and chloral in small doses allay the irritability of the nervous system, on which the condition mainly depends. Faradisation of the pneumogastric is sometimes useful.

**VI. and VII. Diseases of the pharynx (*ante*) and of the nose (*post*) are generally attended by a certain amount of hoarseness and alteration of the voice.** The latter give to the voice a peculiar nasal twang which is very characteristic.

### The Nasal Cavities.

§ 131. *SYMPTOMS and PHYSICAL EXAMINATION.* Diseases of the nose will be considered under three cardinal SYMPTOMS:—*Inodorous discharge* from the nose (Rhinorrhœa); *foul discharge* from the nose (Ozæna); *mouth-breathing* and snoring (Obstruction of one or both nostrils). *Bleeding* from the nose also occurs in some nasal disorders, but it is *not* a cardinal symptom. It is perhaps more generally associated with some constitutional or general derangement. *Sneezing*, *tickling* in the nose, and *sniffing* may also be present; and the quality of the *voice* may be altered, particularly in nasal obstruction. The sense of *smell* is always disturbed to some extent in nasal disorders. In some instances, headache, vertigo and other nervous derangements are met with in association with disorders of the nose, especially when the free transit of air through the nasal passages is interfered with, and the atmospheric pressure within the tympanum disturbed.<sup>1</sup> Various constitutional symptoms may result from septic conditions of the nose or the adjacent sinuses; and not infrequently a patient suffers from listlessness and general debility for a long time before our attention is directed to the true source of his troubles.



FIG. 60.—  
NASAL  
SPECULUM.

*CLINICAL INVESTIGATION.* Rhinoscopy or examination of the nose may be effected through the anterior nares (anterior rhinoscopy), and the posterior nares (posterior rhinoscopy); and by digital examination posteriorly.

**ANTERIOR RHINOSCOPY.** First examine the anterior nares for any obvious disorder, such as fissures, ulcers, scars from ulcers, any narrowing of the nares, or a deviation of the septum; secondly, introduce a speculum (Fig. 60), using either a direct light or one reflected from a mirror on the forehead as in laryngoscopy. In this way an examination of the

<sup>1</sup> A notable instance in my own experience was that of a lady of 35 who suffered from most troublesome tinnitus aurium and occasional giddiness, which was not relieved until the middle turbinate bone was removed by Dr. Scanes Spicer (see the Author's "Clinical Lectures on Neurasthenia," 2nd edition).



inferior turbinate bone can be made, to see if it be hypertrophied. The inferior and middle meati can be thus examined for polypi or alteration in the mucous membrane. If, as frequently happens, the anterior part of the inferior turbinate is hypertrophied, and hides the view, this may be reduced by swabbing out with a cotton-wool pledget soaked in a 10 per cent. solution of cocaine.

POSTERIOR RHINOSCOPY is effected by precisely the same procedure as in laryngoscopy (§ 121), using the smallest of the mirrors and turning it upwards. It is convenient to have a special mirror for this purpose mounted on a curved handle, the stem being hinged at its extremity, so that it can be turned to any desired angle. It is important to avoid touching either the dorsum of the tongue or the posterior wall of the pharynx. The patient should be instructed to breathe gently all the while through the nose. By moving the mirror slightly in different directions we are able to examine the posterior nares, and turbinated bones, the inner end of the Eustachian tube for any swelling, and Luschka's tonsil (*cf.* Fig. 50). The pharyngeal or Luschka's tonsil is a mass of lymphoid tissue on the pharyngeal roof and posterior wall above and between the Eustachian tubes; when in a condition of hyperplasia it forms the cushion-like growth of post-nasal adenoids (§ 112).

A great deal of information may be derived by passing the finger behind the soft palate, but for this purpose it is generally necessary to spray the pharynx with cocaine (10 per cent.).

Our *first* inquiries concerning any given case of suspected disease of the nose should be relative to the LEADING SYMPTOM, especially whether there be any nasal discharge, and whether it is inodorous or foul-smelling. We cannot depend upon the patient's statement on this point, because very often the same disease which causes a foul discharge may also blunt the sense of smell. *Secondly*, we must investigate the HISTORY and whether any of the other symptoms above mentioned were present. *Thirdly*, we must proceed to the PHYSICAL EXAMINATION by testing whether the patient can breathe freely through each nostril separately; by examining the anterior, and, if necessary, the posterior nares.

**Classification.** Diseases of the nose, like those of the throat, are best classified by the PHYSICAL SIGNS met with on examination—

viz., **nasal discharge, nasal obstruction, epistaxis**—and their **causes**. Thus :—

a. **ACUTE INODOROUS DISCHARGES** (Acute Rhinorrhœa)—the causes of which are—

I. Acute rhinitis; II. Syphilis (snuffles); III. Diphtheria and other fevers; IV. Coryza; V. Hay Fever; VI. Glanders.

b. **CHRONIC INODOROUS DISCHARGES** (Chronic Rhinorrhœa)—the causes of which are—

I. Chronic simple rhinitis; II. Chronic hypertrophic rhinitis; III. Cerebro-spinal rhinorrhœa; IV. Ulcerations of the nose, polypi, and occasionally catarrh of the sinuses.

c. **CHRONIC OFFENSIVE DISCHARGES** (Ozœna), which have for causes—

I. Ulcerations and bone disease—Syphilis, tubercle, and lupus; II. Atrophic rhinitis; III. Empyema of antrum and other sinuses; IV. New growths and polypi breaking down, and impacted foreign body.

d. **NASAL OBSTRUCTION** (Snoring, and mouth-breathing)—the causes of which are :

I. Pharyngeal adenoids; II. Polypi; III. Deviated septum; IV. Hypertrophy of turbinate; and V. Foreign body and neoplasms in adjacent parts.

e. **EPISTAXIS**, the causes of which may be Local or General.

§ 132. **Acute (or recent) inodorous discharge from the nose (Rhinorrhœa).** *Discharge is a frequent symptom when disease of the nose is present, and we should endeavour to ascertain if this be odourless or offensive, although these are, of course, only relative terms, and the two groups cannot be sharply defined. Among the causes of ACUTE INODOROUS DISCHARGE, congenital SYPHILIS should be suspected in infancy; DIPHTHERIA in childhood; CORYZA in adults.*

I. **Acute Rhinitis** may be set up by *irritation* of any kind, as the vapour or dust of some trade; or by any injury. For instance, a profuse discharge from one nostril in a child should always make us suspicious of his having inserted a pea, marble, or other *foreign body*, although the history may be wanting. But its commonest cause is “a cold” (see Acute Coryza, below).

II. **“The Snuffles.”** In newly-born children, congenital syphilis is almost invariably attended by a profuse nasal catarrh, and is known familiarly as “the Snuffles.” The other features of nasal syphilis will be referred to under Ulcerations.

III. **Diphtheria** and other Fevers. In diphtheria a profuse nasal discharge excoriating the upper lip with slight elevation of

temperature, and prostration, coming on suddenly in a child or young person previously healthy, is so characteristic that the disease may be almost diagnosed from these features alone.

IV. In **Acute coryza**, "catarrh," or "cold in the head," there is profuse muco-purulent discharge attended by sneezing, running from the eyes, and febrile symptoms with frontal headache, extending over a few days. It is usually attributed to some exposure to cold ("a chill"); but it frequently prevails in an epidemic form, and is then probably of microbic origin. It is predisposed to by cold and damp weather, by adenoids and the other causes of chronic rhinitis. It is not a serious disorder, but its repeated occurrence may lead to middle ear catarrh, or to bronchitis by extension.

*Treatment of "catarrh."* In severe cases, it is advisable for the patient to keep in bed. At the outset a full dose of Dover's powder given at night, or a mixture of tr. aconit. ℞j, liq. ammon. acetat., with other salines, every two hours, may cut short the disease. Locally, sprays of cocaine (2 to 4 per cent., applied with caution, occasionally), or equal parts of boracic acid and borax dissolved in water, or camphor and menthol (gr. 8 to the oz. of paroline), may abort the disease. Ferrier's snuff<sup>1</sup> is also useful. Inhalations of camphor, menthol or vinegar, taken at night, are reputed to be efficacious.

V. **Hay fever**, or as it is sometimes called, hay asthma, is a severe catarrh of the nasal mucous membrane and conjunctivæ, coming on pretty regularly in the summer or autumn of each year, presumably connected with the pollen of flowers. It is accompanied by the symptoms of severe Coryza just described, which come on somewhat suddenly in a person predisposed, who has been outdoors (usually in the hay-making season), and are attended by a certain amount of constitutional disturbance. There appear to be two clinical varieties of this disease: (i.) where the symptoms are chiefly constitutional: and (ii.) where the symptoms are chiefly local. In the latter there is generally hypertrophy of the inferior turbinate, which constitutes an important predisposing factor.

The *etiology* of this disease is somewhat obscure, but it evidently is connected in some way with the pollen, especially of grasses. There are some people, usually those with a marked neurotic taint, who cannot go within a couple of miles of a hay-field in the summer without developing the disease. It is diagnosed from simple coryza chiefly by its seasonal occurrence. It resembles asthma in some respects, especially in its periodicity, differing in that the nasal, instead of the bronchial, mucous membrane is involved. The malady is not a fatal one, but causes serious

<sup>1</sup> Bismuth Subnitrate 5vi; Morph. Hydrochlor. gr. ij.; Pulv. Acac. 5ij.

discomfort and inconvenience. Sometimes people get rid of it as they get older, but in some it continues throughout life.

*Treatment.* The first indication is the avoidance of the cause. This may be accomplished by a sea voyage, residence at the seaside at a high altitude, or by living indoors in the city, taking care that no plants or flowers enter the house. But there is no rule in this respect; for some do better at a high altitude, others at a low one; some get better at the seaside, others in a town. Quinine, arsenic, iron or belladonna may be taken before the attack is expected. Antipyrin (gr. 15) has been credited with cutting short an attack. If the disease extends to the bronchi, asthma papers and cigarettes should be employed. Locally, means should be taken to prevent the pollen reaching the mucous membrane. For this purpose Brunton recommends smearing the nostrils with zinc oxide ointment, which not only allays the irritation, but by remaining longer unmelted is more efficacious than other ointments. Antiseptic sprays destroy the pollen; of these quinine, gr.  $\frac{1}{2}$  to the oz., dissolved in normal saline solution, as being less irritating than water, gives good results. Sir Andrew Clark recommended swabbing out with hyd. perchlor. gr. i, quin. hydrochlor. gr. ii, glyc. ac. carbol. 2 oz. To relieve the discomfort, cocaine tabloids (gr.  $\frac{1}{4}$ ) inserted in the nose, sprays of cocaine (4 per cent.) or menthol (20 per cent.), or silver nitrate (gr. 10 to the oz.) are used. The mucous membrane, if thickened, must be treated as in Hypertrophic Rhinitis.

**VI. Glanders.** The copious discharge of viscid semi-purulent matter from the nostrils is one of the earliest symptoms of Farcy, or Chronic Glanders (Chap. XV.).

§ 133. In **Chronic nasal discharges** it is still more difficult to draw the line between odorous and inodorous discharges, since many of the conditions, though odourless at the outset, become offensive later on; and it will generally be necessary to pass in review all the conditions mentioned in this section and § 134 below. The following are the chief causes of INODOROUS DISCHARGE:—

**I. Chronic Rhinitis** is a chronic inflammatory condition of the mucous membrane of the nose, attended by increased secretion, and usually by thickening. It occurs in three forms: (a) SIMPLE; (b) HYPERTROPHIC (*infra*); (c) ATROPHIC (§ 134). The first two give rise to an *inodorous*, but the ATROPHIC to an *odorous* discharge.

CHRONIC SIMPLE RHINITIS consists of a chronic congested, and sometimes, later on, a hypertrophied state of the mucous lining of the nose, attended by a continuous mucous or muco-purulent discharge. There is generally a certain amount of nasal obstruction, giving rise to altered voice and snoring.

*Etiology.* (i.) It is predisposed to by cardiac and pulmonary disease, alcoholism, and the strumous diathesis. It may be



determined by (ii.) recurrent attacks of neglected coryza over a long period of time; (iii.) the injury caused by an unsuspected foreign body, in which case the condition is generally confined to one side; or (iv.) the constant irritation of dust and noxious vapours—*e.g.*, masons, fustian-cutters. (v.) It is often associated with adenoids, enlarged tonsils, and other causes of obstruction to the nasal respiration.

*Prognosis.* The disease is essentially a chronic one, and requires prolonged treatment. The chief fear, and what in point of fact frequently happens, is that middle ear catarrh may result from the extension of the inflammation up the Eustachian tube. Even apart from this, it is very important to treat these cases in strumous children, because the condition interferes with the respiratory functions of the body.

*Treatment.* In the early stages alkaline washes, sod. bicarb. gr. xv, and borax gr. v, or acid carbol. gr. iij to the oz., sniffed up or given by the nasal douche.<sup>1</sup> This is followed later on by a spray of menthol and eucalyptol (gr. 30 to the oz. of aquol or parelein), or an ointment of cocaine and thymol (gr. 10 to the oz. of white vaseline), or by the use of the ammonium chloride inhaler. Constitutional treatment is necessary, by means of tonics, cod-liver oil and malt. Alcohol should be avoided, and a high and dry climate should be sought. In the later stages, the only satisfactory method of treatment is applying chromic acid (gr. v or x to ʒj), or still better the galvano-cautery.

II. **Chronic hypertrophic rhinitis** is a special form distinguished from the preceding by the fact that there is considerable hyperplasia of the nasal mucous membrane, especially over the inferior turbinate bone at its anterior and posterior ends. It presents the same symptoms as the preceding, only in a greater degree. It is apt to occur in neurotic people, and even in slight cases to be accompanied by headache and mental depression. It is frequently associated with adenoids. The *prognosis* is on the whole less favourable. The *treatment* is much the same, but more active measures are indicated, and especially treatment by the thermo-cautery.

<sup>1</sup> A nasal douche is always preferable, but it requires practice for its use. A very imperfect substitute is to fill the palm of the hand with the liquid and sniff it up the nose.

III. **Cerebro-spinal Rhinorrhœa.** Dr. St. Clair Thomson<sup>1</sup> has shown that the cerebro-spinal fluid sometimes escapes from the skull, probably through the perineural sheaths of the olfactory nerves, and probably owing to some pathological change leading to increased intracranial pressure. It is *recognised* by :—(1) The constant and long-continued escape from the nostril of a perfectly clear watery fluid. This alone should arouse our suspicion. (2) The fluid has no odour, taste, nor sediment; is free from albumin and mucin, and reduces Fehling's solution. This last is absolutely distinctive.

IV. **Ulcerations of the nose, Polypi, Disease of the sinuses,** occasionally produce inodorous discharges; but the discharge is more often offensive (see below). **CHRONIC FRONTAL SINUS EMPYEMA** is, however, attended by (i.) a purulent, *non-fetid* nasal discharge,<sup>2</sup> (ii.) frontal or supra-orbital headache or feelings of discomfort, and (iii.) more or less well-marked nasal obstruction, caused by inflammatory enlargement of the middle turbinated body, or by polypi. Headache only occurs from retention, and not when drainage is free. There may be tenderness on pressure over the affected side.

§ 134. **Ozœna** or a **chronic offensive discharge from the nose** may occur in the later stages of MANY OF THE CONDITIONS mentioned in the preceding section. But the chief causes of foul discharge from the nose are as follows, and the commonest and foulest of these are **SYPHILITIC DISEASE** in middle life, **CANCER** in the aged.

Foreign bodies, which have already been referred to, and Polypi, will be described under **Nasal Obstruction** (§ 135), which is their leading symptom. It will be necessary to give some detailed account of—Ulcerations (and Bone disease); Atrophic Rhinitis; and Empyema of the Sinuses.

I. **Ulcerations and bone disease** attacking the nose are mostly of syphilitic, occasionally of tubercular, origin. Neoplasms in the later stages ulcerate, but in the earlier stages give rise to Rhinitis or Nasal Obstruction (§ 135).

(a) **Syphilitic rhinitis.** In the early stages of syphilitic infection we may get an acute catarrh with superficial ulceration, which is the condition found in children with congenital syphilis, known as "snuffles." In the later stages gummata form in various situations, which *rapidly involve the bone* and other parts; the discharge then becomes very foul. The ulcers have the same character as those affecting the throat (*q.v.*).

(b) **Tubercular ulceration** more often involves that part of

<sup>1</sup> "The Cerebro-spinal Fluid: Its spontaneous escape from the nose," by St. Clair Thomson. Cassell and Co., London, 1890. And see also *B. M. J.*, Sept. 23, 1892, p. 794.

<sup>2</sup> Dr. Herbert Tilley, *The Lancet*, July 14th, 1900.

the nose near the orifice, but otherwise the ulcers much resemble the preceding. They are differentiated from them by their very much slower progress, as well as by their site. The bones are rarely attacked, and consequently the discharge may be more or less inodorous; and there is rarely the falling in of the bridge of the nose, which so frequently occurs in tertiary syphilis. The ulceration of *Lupus* differs but little from the true tubercular ulceration, excepting that *lupus vulgaris* usually involves also the skin of the *alæ nasi*, whence it has probably spread.

Atrophic rhinitis is distinguished from these ulcerations by the pallor and thinning of the mucous membrane, the absence of visible ulcers, and the absence of a history or evidences of syphilis or tubercle respectively.

The *prognosis* of nasal ulceration is fairly good if the patient come under treatment early, but if not it leads to considerable destruction of tissue. Tubercular ulceration may slowly lead to the destruction of the *alæ* of the nose, but syphilis results in the most extensive destruction of the *bones* both of the septum and the palate; the bridge of the nose falls in, and the anterior nares may be represented by a single gaping orifice. It is this extensive and rapid destruction which is so pathognomonic of nasal syphilis.

The *treatment* should be much more prompt and vigorous in ulceration of the nose than in chronic rhinitis and similar affections, because of the destruction which ensues. Carbolic and astringent sprays are useful palliatives, but surgical measures are called for if the bone is involved. All dead bone must be removed. Tubercular ulcers should be scraped. Large doses of pot. iodide lead to rapid healing of syphilitic ulcerations.

II. **Atrophic rhinitis**, also known as idiopathic or true *ozæna*, is characterised by (i.) a thick foul discharge, which is sometimes profuse, sometimes scanty: (ii.) the nasal cavities are often large, and the bridge of the nose broad and sometimes depressed. The mucous membrane is thin, pale, and covered with crusts, hard, adherent, and decomposing. Sometimes it is unilateral, *e.g.*, in cases of deviated septum. A certain amount of chronic pharyngitis is usually present. (iii.) The breath has a foul odour, which is not detected by the patient, as the sense of smell is blunted. It is *diagnosed* from the other causes of *ozæna* by the absence of ulceration, the presence of atrophied mucous membrane, and wide cavities.

*Etiology.* (i.) It is commoner in the young and in women. It usually starts before 16 years of age. (ii.) Unilateral atrophic rhinitis is mostly due to some local cause, such as a deviated septum or sinus disease, the

narrower side being healthy. (iii.) The exciting causes of bilateral atrophic rhinitis are obscure : it has been said to follow chronic rhinitis in strumous children ; (iv.) in some cases it is a sequence of hypertrophic rhinitis.

*Prognosis.* Prolonged treatment is necessary for its cure, and even this is not very hopeful if the disease be advanced. The disorder is generally most marked at about 20 years of age ; it becomes less troublesome at middle age, and as it gradually disappears with advancing years we may presume that it tends slowly to spontaneous cure.

*Treatment.* Alkaline and antiseptic douches and sprays are indicated, as in § 133. To stimulate the mucous membrane nasal tampons of cotton wool soaked in glycerine are used. These are useful in unilateral rhinitis, as they ensure respiration through the narrower cavity. The nose may be swabbed out with silver nitrate (gr. 10 to oz.), or with trichloroacetic acid (5 to 20 parts in the 1000), which removes the smell. Constitutional treatment is also advisable.

III. **Chronic Empyema of the antrum** and other sinuses is a term applied to a chronic suppurative inflammation of the lining membrane ; though the term empyema should be reserved for cases in which there is retention. The most constant and cardinal symptom is a purulent or sero-purulent discharge from *one nostril*, which is generally offensive or sickly. It may arise as an extension of nasal catarrh, or various suppurative nasal conditions (syphilis, tubercle, bone disease, etc.). A probe cannot be introduced if the orifice communicating with the nasal cavity is blocked.

*Empyema of the antrum* may be due to irritation from a tooth. Many of the patients have had decayed teeth in the upper jaw. It is recognised by the discharge being intermittent, returning usually about the same time each day, and flowing freely when the head lies on the opposite side, or is lowered between the knees. The discharge may be seen coming from beneath the middle turbinate. If a bright light is held in the mouth the cheek of the affected side remains darker than the other (trans-illumination).

Discharge from the *frontal* or *anterior ethmoidal* sinuses flows best when the patient is upright. It comes from under the middle turbinate, and there is often pain in the brow and orbit. See also p. 258.

Discharge from the *posterior ethmoidal* and *sphenoidal* sinuses flows over the middle turbinate and down into the pharynx. There may be exophthalmos, ptosis, strabismus, etc., with disease in this locality.

Various constitutional symptoms have recently been recognised as being associated with Sinus Disease, due probably to the toxæmia which results from septic absorption.<sup>1</sup> Lassitude, headache, occasional elevations of temperature, and numerous nervous and vasomotor symptoms are among the commonest. They generally present a periodic or paroxysmal character. Trifacial neuralgia may also result from sinus disease. If overlooked or neglected empyema may excite middle ear catarrh (with tinnitus, deafness, etc.), recurrent nasal catarrh, and nasal polypi.

*Prognosis and Treatment.* Sinus empyema is chronic and intractable, but very rarely fatal. The treatment is based on surgical principles, but the chief indications are—free drainage, stimulation of the chronic inflammation until it takes on a more healthy action of repair, or scraping.

IV. **Neoplasms and Polyphi** (§ 135) and **Impacted foreign body** (§ 132—I.) are referred to elsewhere.

<sup>1</sup> See also "A Treatise on Nasal Suppuration," by Dr. Ludwig Grünwald. Trans. by Wm. Lamb. London, 1900.



**§ 135. Nasal obstruction, snoring and mouth-breathing.**

*Nasal obstruction may be partial or complete, and it may exist on one or both sides. It is met with in a greater or less degree in nearly all of the various nasal conditions previously discussed, and it is a marked feature in HYPERTROPHIC RHINITIS, p. 257. Its commonest cause in children is PHARYNGEAL ADENOIDS (p. 229). It is also a cardinal symptom in NASAL POLYPI, DEVIATION OR SPUR OF THE SEPTUM, ALAR COLLAPSE, FOREIGN BODIES, NEOPLASMS, and ABSCESSSES.*

*Effects.* Apart from the inconvenience of snoring, nasal obstruction renders the individual prone to pharyngitis, stomatitis, bronchial catarrh, and other consequences due to the entry of cold air into the lungs without being properly warmed by its passage through the nose. Among the other consequences are, a nasal quality of the voice, distortion of the chest (when arising early in life), and impeded respiratory functions of the body generally. These disorders consequently assume an importance quite out of proportion to the degree of local mischief.

I. **Pharyngeal adenoids** are of very frequent occurrence. They constitute one of the forms of granular pharyngitis, and the disease has been referred to under that condition (§ 112). It is the most frequent cause of mouth-breathing and snoring in children. It is often overlooked by parents, a circumstance greatly to be regretted for three reasons. In the first place, it is one of the most potent causes of chronic otitis media and deafness in after life; secondly, it impairs the respiratory functions of the body, as just mentioned; and thirdly, the open mouth and vacant aspect which are so characteristic produce an appearance of backward intelligence, which in point of fact sometimes also exists.<sup>1</sup>

II. **Polypi**, or pedunculated tumours, are the most frequent new growths in the nose. Polypi are of three kinds, a. GELATINOUS; b. FIBROUS; and c. MALIGNANT.

a. GELATINOUS OR MUCOUS POLYPI are the most common form of polypi. They usually consist of myxomatous tissue, and are generally associated with inflammatory disease of the subjacent

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<sup>1</sup> This result is not altogether explicable by faulty respiratory processes of the body; nevertheless I am satisfied that the intelligence in some children has been dull until the pharyngeal defect has been remedied.

bone. They are often multiple, and most often grow from the mucoperiosteum of the upper and middle turbinated bones. Their detection is not difficult, for in addition to the feeling of "stiffness" and the watery discharge (which may be intermittent), they are easily seen through the nasal speculum as pale grey glistening bodies. They are apt to recur after removal, but are not malignant in other respects.

b. **FIBROUS POLYPI** grow from the roof of the naso-pharynx. By their growth they displace the parts around and are apt to give rise to "frog-face." The discharge is often foul and may be hæmorrhagic. They sometimes become malignant (fibro-sarcoma). They may occur at any age.

c. **MALIGNANT POLYPI** may be either fibro-sarcomatous or carcinomatous. They are known by their rapid growth, and the resulting deformity of the face, "frog-face," and by the offensive and hæmorrhagic discharge. Sarcomatous growths are chiefly met with in the young; carcinomatous in the aged.

*Prognosis and treatment.* The benign polypi are not dangerous to life, but are liable to recur. Malignant growths give rise to a condition of considerable gravity. Occasionally fibrous tumours atrophy. Gelatinous polypi, arising as they do from the anterior part of the cavity, can generally be removed by means of a nasal snare or forceps; but the other varieties, springing usually from the posterior parts and infiltrating the tissues around, may require an operation of some magnitude.

**III. Deviated septum and nasal spur.** The nasal septum is rarely quite in the median line, but the displacement is often considerable. Sometimes it results from injury. Various consequences may ensue, such as hypertrophied turbinate on one side, atrophic rhinitis on the other. When an angle is formed in the septum nasi it is spoken of as a "spur," and this is most readily dealt with by sawing it off.

**IV. Hypertrophied Turbinate** is met with usually either as part of, or a consequence of, chronic hypertrophic rhinitis. It may occur on one or both sides, and in either case, in narrow nostrils, produces partial obstruction, snoring, and mouth-breathing. It is removable by turbinectomy: sometimes the thermo-cautery is employed (see Hypertrophic Rhinitis, p. 257).

**V. Foreign bodies** within the nose. **Neoplasms** and **Abscesses** in adjacent parts may also produce *unilateral* nasal obstruction.

§ 136. **Epistaxis** (bleeding from the nose) may be a symptom of nasal disorders; but if in any appreciable quantity it is usually

an evidence of some general disorder. Not infrequently both general and local causes are in operation, and the *nasal cavities should always be carefully examined*. The blood-vessels give way in this situation (sometimes as a kind of safety-valve) merely because they are thin-walled, numerous, and near the surface. So much is this the case that the diminished atmospheric pressure to which mountaineers are subjected is sufficient to produce nose bleeding, when they reach great heights. The *causes* may be divided into two groups—Local and Constitutional.

a. LOCAL CAUSES, in which the hæmorrhage consists usually of little more than streaks, may arise from any marked congestion of the mucous membranes, such as that which accompanies adenoids, acute rhinitis, or worms in the nose;<sup>1</sup> or as a consequence of mechanical violence, applied either directly to the nose, or to the base of the skull. Any serious destructive disorder—such as new growths, especially malignant, syphilitic, tubercular, or other ulcerations (which if small are *very apt to be overlooked*)—may be attended by a certain amount of recurrent bleeding. In these circumstances the hæmorrhage is usually an intermittent and subordinate feature. The diagnosis rests on the characters already given. When small in quantity the blood often passes backwards into the throat, and is swallowed; or it may be expectorated or coughed up, and be mistaken for hæmatemesis or hæmoptysis.

b. With CONSTITUTIONAL CAUSES the bleeding is usually, although not always, of larger quantity, and it may indeed be so profuse as to endanger life. The blood in this group comes from a spot near the anterior part of the septum. Among the *predisposing causes* none is more frequent than an idiopathic tendency which exists in certain individuals to bleed upon slight provocation, a tendency which runs in families. Without amounting to hæmophilia, certain persons undoubtedly present some inherent quality which renders them more liable to bleed from their mucous surfaces, with or without a wound. It may exist in only one member of a family, but more often in several brothers and sisters. I have often noticed that such a

<sup>1</sup> Dr. Manassch relates the case of a child with epistaxis in whom a leech was found in the nose. This had gained entrance by the child drinking at springs in a district where leeches abounded in the water. *The Lancet*, Sept. 16, 1899, p. 785.

predisposition may exhibit the phenomenon of atavism and skip a generation. Epistaxis is more frequent in children, especially in boys. It is also met with in the aged, but only when vascular disease and some of the other conditions about to be mentioned exist. The constitutional causes may be grouped under (a) *alterations in the cardio-vascular system*, and (b) *altered blood states*.

a. Epistaxis occurring for the first time in an apparently healthy person over 40 years of age, should always give rise to the suspicion of Chronic Bright's Disease. It affords us, moreover, an indication for the treatment of this malady of which advantage may sometimes be taken, for it relieves the vascular tension which would otherwise seek relief in some less favourable situation. For instance, I have observed several patients who, after repeated admissions to hospital for epistaxis, have finally come in to die of cerebral hæmorrhage. Epistaxis is a frequent consequence of cardiac valvular disease, emphysema, chronic bronchitis, and cirrhosis of the liver. It may also be an evidence of lardaceous, or other, disease of the vessels. Finally, epistaxis is one of the forms of vicarious menstruation; and like the bleeding which may take place in hysteria and other conditions where the vaso-motor system is disordered, we must regard this as an extreme effect of disease of the sympathetic nervous system.

b. Concerning *altered blood states*, it may occur with purpura, hæmophilia, scurvy, leukæmia, anæmia (simple, and especially pernicious), and the specific fevers. It is in children a not infrequent prodromal manifestation of whooping-cough and similar microbic disorders.

*Prognosis.* Slight epistaxis in children is of no consequence, but occurring for the first time in persons at or past middle life, it should receive our serious attention, and its cause be carefully investigated. Inquiry should always be made as to whether it has occurred previously in the life of the individual, because, as above mentioned, certain persons seem to have this tendency, and in these the symptom is not of much importance.

*Treatment.* The indications are—first, to check the hæmorrhage if profuse, and secondly, to ascertain the cause.

The epistaxis which accompanies Bright's disease, and the congestion of cardiac and pulmonary disease should not be checked



unless the amount be profuse. In such cases the epistaxis is usually preceded by headache and is accompanied by high arterial tension. It may be one of Nature's methods for the relief of congestion, as evidenced by the fact that the headache and the high arterial tension are relieved by the hæmorrhage. In all cases of epistaxis, the first thing to do is to examine the arterial tension; so long as this remains high or moderate no harm can accrue from the epistaxis.

(a) The treatment of *the attack* resolves itself into checking the hæmorrhage. The patient should be kept perfectly quiet, with the head erect, and chin forward, the head being cool, the feet warm, with hot bottles if necessary. The arms may be raised above the head and ice applied to the lower cervical spine. A homely substitute for the latter has long been in vogue in the form of the front door key. Some recommend pressure to the anterior part of the septum by the thumb and forefinger externally. More extreme measures consist of the application of ferri perchlor. to the site of the hæmorrhage, if this can be discovered, or the use of styptic sprays, of hamamelis, catechu, vinegar, lemon-juice, etc. Finally, if all these fail, the posterior nares must be plugged by means of Belloc's canula, or a simple makeshift in the form of a loop of malleable wire.

(b) *Between the attacks*, a very thorough investigation of the nasal and post-nasal cavities must be made. Minute lesions, quite sufficient for epistaxis, are very easily overlooked. The treatment of recurrent epistaxis is not always an easy matter, for the cause is often obscure, and we are often driven to regard the case as belonging to the idiopathic group above referred to. In a good many cases iron is efficacious in warding off the attacks; and calcium chloride by increasing coagulability.

## THE THYROID GLAND.

This gland is anatomically connected with the upper respiratory passages, but is physiologically quite separate. There is still much for us to learn concerning its functions, both in health and disease. In health it is concerned in what is called "internal secretion," and it either adds to or modifies the blood in some way that is necessary

to the well-being of the individual. This is proved experimentally,<sup>1</sup> and is evident from the gravity of the symptoms which arise when the gland is diseased, or is removed by operation.

**SYMPTOMATOLOGY.**—There are two opposite clinical conditions which may arise from disease of the thyroid gland. In one there is a *diminished* thyroid action, a condition of *Athyroidism*, the symptoms of which (lethargy, lowered vitality, and impaired growth and development) are identical with those of Myxœdema. These effects also arise, as Horsley (*loc. cit.*) has shown, when the gland is extirpated. The other condition is one of *increased* thyroid action or *Thyroidism*, the symptoms of which (neuro-vascular irritation) resemble Graves' disease; and these, with the exception of the proptosis, can be produced by the internal administration of thyroid gland or extract in large doses. It is important to remember that the size of the gland is not always a guide to which of these two sets of symptoms we may expect in any given case; for enlargement of the gland is consistent with atrophy of the glandular elements and diminution of function; and *vice versâ*. It depends on the nature of the histological changes, and these need further elucidation.<sup>2</sup>

§ 137. **PHYSICAL EXAMINATION and CLASSIFICATION.**—There are but two physical signs referable to the thyroid gland, viz., enlargement and diminution of volume. The size of the gland can never be gauged with precision, and when the alteration of volume is only slight it is difficult, if not impossible, to estimate it with accuracy, because it is partially covered by muscles, and so intimately connected with the trachea and other deeper structures. The patient should be instructed to let his head fall forwards and *to swallow* whilst we endeavour to gently grasp the gland. The thyroid rises during deglutition as no other neck tumour or organ does. Some idea may be obtained of the progress of a case by measuring the neck from time to time, always exactly at the same level.

**Classification.** In general, *enlargement* is attended by a

<sup>1</sup> Sir Victor Horsley, *Clinical Journal*, March 8th, 1899, and Trans. Roy. Soc.

<sup>2</sup> True hypertrophy, with increased functioning of the thyroid gland, is indicated microscopically by great diminution of the colloid material and increase in the secreting cells which line the vesicles. See also Victor Horsley, *Clinical Journal*, 1899, and Trans. Roy. Soc. in the previous years.

condition of thyroidism (*e.g.*, Graves' Disease), and a *diminution* by a condition of athyroidism (*e.g.*, myxœdema); and there are two well-marked type of disease which are *usually* associated with enlargement, and two with diminution in volume of the thyroid gland.

(a) The two diseases (besides cancer and other neoplasms) in which **enlargement of the thyroid** is—at some stage of the malady—the essential or pathognomonic feature<sup>1</sup> are :—

I. GRAVES' DISEASE—or Exophthalmic Goitre—is the term applied to that form of enlargement of the thyroid which, coming on in adult life, is attended by proptosis, and by numerous cardio-vascular and nervous symptoms, with marked disturbance of the general health. These general symptoms collectively constitute THYROIDISM. They are often present long before there is any visible enlargement.

II. SIMPLE GOITRE or “BRONCHOCELE” is the term applied to a simple increase in size of the thyroid gland, either congenital or coming on in childhood or early adult life, generally unattended by increase or diminution of function, and unaccompanied, therefore, by any symptoms other than the mechanical effects of enlargement. In other words, there is in most cases a condition neither of thyroidism nor athyroidism.

IIa. BRONCHOCELE OF ADOLESCENCE is the name under which I propose to include the slight, usually tender, but painless enlargements, which are not uncommon in young women, transitory (a few weeks) in duration, probably inflammatory in origin, and usually unattended by any general symptoms.

(b) There are also two diseases in which **atrophy of the thyroid**—or at any rate a diminution of its function (and usually of its size)—is the essential feature of the malady :—

I. CRETINISM is the term applied to the condition of stunted growth (both in mind and body) of the individual, due to congenital atrophy or absence of the gland function, although the gland itself may be either enlarged or diminished in size. It is a condition of congenital ATHYROIDISM. The disease is endemic in certain districts.

II. MYXŒDEMA is the term applied to the group of symptoms (lethargy, low vitality, etc.) which, coming on in adult life,

<sup>1</sup> In ACROMEGALY (Chap. XVII.) the thyroid is sometimes slightly enlarged or diminished in size, but it has generally been regarded as a subordinate feature in this rare and strange disease. In some cases of CRETINISM (which is included in group b) the thyroid gland is considerably enlarged, but *deficient* thyroid action constitutes the essence of the disease.

accompany *atrophy* of the thyroid gland. This is a condition of acquired **ATHYROIDISM**, or sporadic adult cretinism.

It is legitimate to infer from the foregoing that—

1. Increased or disordered thyroid secretion gives rise to profound disturbance of the general health, and neuro-vascular irritation (Graves' disease).

2. An innocent enlargement of the thyroid, unaccompanied by increased or disordered thyroid secretion, has no effect on the economy (as in most cases of bronchocele).

3. Simple absence or diminution of the thyroid secretion results (*a*) when it is congenital or comes on in early life, in deficient development, mental and physical (*i.e.* cretinism); and (*b*) when it supervenes in adult life, in lethargy and deficient vitality (myxœdema).

**§ 138. Graves' Disease** (Synon.: Exophthalmic Goitre, Basedow's disease) has been defined on the preceding page. It may occasionally come on suddenly, but usually the onset is very insidious. There are *five* groups of symptoms, and the varieties of the disease depend on which of these predominate.

*Symptoms.* 1. *Cardio-vascular* disturbances are among the earliest and most important symptoms. They are never absent, and may exist for months before any other evidence appears: (i.) Palpitation. (ii.) The increased frequency and tumultuous action of the heart is accompanied by a rapid and sometimes feeble pulse, up to 150 or more on the slightest exertion, or emotion. The rate may sometimes be reduced by absolute rest to 40 or 50. (iii.) Paroxysmal dyspnœa and a distressing sense of suffocation, produced and relieved by the same causes as the preceding. (iv.) Evidences of cardiac disease, such as the murmur of dilatation (said to be present in two-thirds of the cases) (§ 47), are frequently present. The commonest murmur is a systolic, heard loudest over the second left costal cartilage and sometimes propagated up the vessels of the neck. (v.) Sometimes slight dropsy and occasionally albuminuria are observed, as consequences of the ventricular dilatation.

2. *Nervous* disturbances are always present, and very closely resemble the neuro-vascular phenomena of neurasthenia. They are very variable: thus (i.) there may be nervousness, irritability, insomnia, depression alternating with excitement, hysterical attacks, melancholy, or mania. (ii.) Hyperæsthesia, perverted sensations, neuralgic headache, vertigo, tinnitus aurium, and hallucinations of



sight or hearing. (iii.) Other fairly common symptoms are fine and rapid vibratile tremors of the hands, or of the lips (causing stammering); choreiform movements or paresis, usually transient. (iv.) Vaso-motor disturbances of many different kinds, such as "flushings and shiverings"; and various other subjective phenomena. All these symptoms, as well as those in group 1, are characterised, like other symptoms referable to the vaso-motor system, by their paroxysmal character.

3. *Thyroid enlargement* is always present at some stage of the disease, though it is very rarely the first symptom noticed by the patient, probably because there are no means of detecting slight enlargements. Therefore in the early stages we have to rely upon the other symptoms. The enlargement varies considerably in different cases, and is by no means proportionate to the other symptoms; because the symptoms depend more upon the histological element of the gland which is involved than the degree of enlargement (p. 266). Mechanical effects of thyroid enlargement may be present (see Bronchocele); and occasionally alteration in the voice from this cause is the first symptom noticed by the patient.

4. *Exophthalmos*, or protrusion of the eyeballs, is present in a varying degree, though sometimes not until late in the disease (fig. 2, p. 24). It is best detected by seating the patient in a chair, standing behind him and looking down his forehead. As a rule no changes can be detected in the fundi. Later on ulceration of the cornea occasionally takes place, either from neuro-trophic causes or from deficient protection.<sup>1</sup>

5. The *general health* of the patient is always disturbed. Anæmia is pronounced, and is usually in proportion to the severity of the other symptoms. Progressive weakness is always present. The *varieties* of the disease depend on which of these five groups of symptoms predominates.

*Etiology.* (i.) Upwards of 95 per cent. of cases are females. (ii.) Nearly all are young adults between the ages of 15 and 30. (iii.) Locality has no known influence, but Russell Reynolds

<sup>1</sup> Four signs of Graves' disease referable to the eyes bear the names of different physicians. *Abadie's* sign in this disorder is an involuntary twitching or spasm of the levator palpebre superioris. *Von Graefe's* sign is a condition in which the upper eyelid does not follow the eyeball when this makes a downward movement. *Mobius's* sign is an insufficiency of convergence of the two eyes when looking at a near point. *Stellway's* sign is an absence or deficiency of blinking as an involuntary act. All, except the first, are present only in advanced cases, and are not therefore of very great value in the diagnosis.

believed that a low temperature, continuous rainfall, and little sunshine may be determining factors. (iv.) Heredity has not been traced so far as the disease is concerned, but the family often show neuroses in the shape of epilepsy, chorea, hysteria, insanity, etc. (v.) Fright, anxiety, and mental overwork are potent factors in determining the disease.

*Diagnosis.* The four cardinal symptoms are: (i.) Thyroid enlargement, (ii.) Proptosis, (iii.) Rapid cardiac action, (iv.) Nervousness. The early stage of the disease is often mistaken for neurasthenia or for tachycardia.

*Prognosis.* The duration of the disease varies from some six months to many years. It may certainly shorten life, but many very severe cases have recovered under modern methods of treatment. The mortality has been variously stated from 10—50 per cent.; probably more modern statistics would give from only 5—10 per cent. Death may occur from cardiac failure, from some inter-current disease, or with a condition termed Acute Thyroidism, in which there is a great aggravation of the symptoms above described, accompanied perhaps by maniacal excitement.

*Treatment.* The early recognition of the disease is very important, for a great deal can be done in the early stages. Many drugs have been tried. *Iodides* in increasing doses (10 or more grs. ter die) undoubtedly reduce the exophthalmos and thyroid. *Bromides*, especially the ammonium salt, are useful if nervous symptoms predominate. *Digitalis*, *strophanthus* and *belladonna* are very valuable for steadying the heart; by beginning with small doses and persevering, recovery has been effected in some cases. Iron, arsenic, and other nerve tonics are certainly useful. Rest of body, and freedom of the mind from anxiety, are of great importance, combined with other treatment, which must depend on which group of symptoms predominates. Mere sojourn in a hospital will of itself do good, and sea voyages are of use in the same way. One of my cases improved under opium, 4 minims ter die; another under suprarenal extract. Thymus gland extract (10—30 grs. ter die), or  $\text{Zss—j}$  raw gland three or four times a week, has been tried very extensively, sometimes with success.<sup>1</sup>

<sup>1</sup> David Owen, Brit. Med. Assoc. Meeting, *Lancet*, August 22, 1896. Also *E. M. J.* January 7, 1899.—Article by W. Rushton Parker, M.A., M.D., Cantab.

Extirpation of the thyroid<sup>1</sup> has been successfully adopted as a remedial measure, and is indicated when that structure is greatly enlarged. Excision of part of the diseased gland, when very large; and division of the isthmus or excision of the sympathetic cervical ganglia, in cases where the gland is not much enlarged, has been recommended.<sup>2</sup>

Many different forms of ELECTRICITY have been tried, some with undoubted advantage; thus, Bligris ("La Progrès Medicales," 1887) claims that daily faradisation or galvanism of the vagus and sympathetic in the neck produces a marked diminution of the thyroid in fifteen days. A large positive pole, 3 in. in diameter, is applied to the nape of the neck, and a small negative electrode,  $\frac{3}{8}$  in. in diameter, to each carotid for 1½ minutes, and afterwards to each eyelid. Lockwell advises galvanism, applying the kathode over the pit of the stomach and the anode over the nape of the neck, using large electrodes with as strong a current as possible (20–50 milliamperes). Others recommend the same procedure with a weaker current for twenty minutes daily. Denton Cardew (*Lancet*, 1891, vol. ii., p. 6) thinks galvanism the best treatment—the anode placed on the nape of the neck just above the 7th cervical vertebra, the kathode being moved up and down each side of the neck along the carotid, using a weak current (2 or 3 milliamperes) for six minutes *ter die*. In my experience, Faradisation increases the growth and the vascular excitement.

§ 139. **Bronchocele** (Simple Goitre) is another form of enlargement of the thyroid gland. It may affect the whole organ, or only one of its lobes or the isthmus. *Anatomically*, the enlargement may be due chiefly to parenchymatous increase, to cystic enlargement, or to a vascular increase. The enlargement may be so great that the organ amounts almost to the size of an infant's head. It is worthy of notice that the healthy gland may slightly enlarge during pregnancy.

The *symptoms* which attend the disease are entirely those due to the mechanical pressure of the tumour, and it is by the absence of the cardiovascular, nervous, and other symptoms that this condition is distinguished from Graves' disease. The chief pressure symptoms are referable to the larynx and trachea. The voice is modified early in the disease, and vertigo, due to pressure on the vessels, may be present. The general health may be good, but the patient is usually somewhat anæmic, otherwise there are usually no symptoms of thyroidism nor of athyroidism. It may be *diagnosed* from other tumours in the neck by the fact that it invariably rises with the larynx during deglutition. The enlargement generally increases steadily, but it is only rarely that there is any danger from tracheal obstruction and asphyxia.

The *etiology* of the condition is not well known. More females are affected, and it sometimes starts during pregnancy or a catamenial period, but it is most prone to start during adolescence between eight years of age and puberty. It never starts after 40. The disease is endemic in certain districts, and these for the most part are valleys which have a calcium or a magnesium and limestone subsoil, together with a large amount of sulphate and carbonate of lime in the drinking water. That it is not entirely due to the last-named condition is shown by the fact that in districts where this permanent hardness of water exists, bronchocele does not occur. Dogs, horses and mules suffer also from the condition.

*Treatment.* The patient should of course leave the district: or if this is impossible, the water used for drinking purposes should be boiled. When these conditions can be fulfilled, recovery may result. As regards remedies,

<sup>1</sup> *Lancet*, 1894, vol. ii., p. 1045; and 1895, vol. i., p. 1077.

<sup>2</sup> *B. M. J.*, 1899, vol. ii., p. 998, Pollard and Lake.

potassium iod. in gradually increasing doses has the greatest reputation ; and in a less degree ergot, arsenic, and the local application of blisters. Inunctions of mercury or iodine paint have been employed with considerable success. Radical cure consists of the removal of the tumour. The operation of tapping and injecting iodine and other remedies has almost fallen into disuse.

*We now turn to the diseases in which the thyroid is usually DIMINISHED in size, viz., I. CRETINISM, II. MYXŒDEMA. The latter is described elsewhere, since the leading symptom is General Debility (Chapter XVI.).*

§ 140. **Cretinism** is a condition of dwarfism and deformity attended by mental imbecility, due to an absence or perversion of the thyroid secretion, endemic in certain districts. In advanced and typical cases the face is characteristically broad and flat, the tongue protrudes between the teeth, the eyes are wide apart, and the head is brachycephalic (*i.e.*, broad transversely). The skin and hair are dry and coarse, and the mental condition is extremely backward. In severe cases the body may be so dwarfed that a person of 20 is the size of a child of 5. The limbs are shortened, the neck stunted, the abdomen prominent. The thyroid may be enlarged, small, or absent (see Figs. 6 and 7, p. 36). Rushton Parker<sup>1</sup> distinguishes three *varieties*, both etiologically and pathologically. In one, the thyroid is embryologically not developed, or very partially developed, the cause being presumably akin to that which brings about any other embryological deficiency, such as acardia, acephalia, etc. In a second, the thyroid undergoes the same changes as in endemic goitre, and doubtless from the same cause, any differences being due to loss of thyroid function. In a third, the thyroid after performing its functions healthily for a time atrophies, doubtless from causes akin to those of adult myxœdema.

*Etiology.* Cretinism is endemic in certain districts, *e.g.*, the valleys of Switzerland. Cases occur also in certain parts of England, especially in the valleys of the Lake District, and elsewhere. Sporadic cases are found in healthy families. The causes are unknown. Some attribute it to consanguinity of the parents, to alcoholism, tubercle, syphilis, or to maternal worry during pregnancy. It is associated with deficient thyroidal function, and hence may be regarded as congenital myxœdema. In slight cases of cretinism, the diagnosis from other forms of mental deficiency may be difficult. The condition of the skin and hair are valuable diagnostic features.

*Prognosis.* The patient may grow up capable of doing light manual work, or may remain an idiot. Under treatment begun early, the child may recover completely, but in other cases, although the body is greatly improved, the mind does not improve in proportion.

*Treatment.* Thyroid extract, beginning with  $\frac{1}{2}$  gr. doses (gr. 5 of raw gland), causes a rapid and remarkable change. The skin becomes soft, the general conformation normal ; and, if the treatment has not been too long delayed, the mind assumes its natural vigour. The patient must *continue* to take the thyroid all his life, or else he will relapse. A case showing the remarkable efficacy of this treatment is figured on p. 36.

<sup>1</sup> "Acquired Cretinism, or Juvenile Myxœdema," *B. M. J.*, May 29th, 1897.



## CHAPTER VIII.

### THE MOUTH, TONGUE, AND GULLET.

#### The Mouth.

(Lips, Breath, Saliva, Teeth, and Gums.)

WE often regret that we cannot investigate the internal organs more thoroughly, but how seldom do we avail ourselves of the instructive information afforded by a thorough examination of the mouth? Many of the indications of syphilis, hereditary or acquired, may be so revealed; several other constitutional conditions produce symptoms in this locality, such as anæmia and lead poisoning; and a good idea of the general condition of the patient can be obtained from a careful inspection of the tongue. Many of the disorders special to the mouth are comprised among the "causes" of Stomatitis. For the diagnosis of these disorders it is necessary to make a thorough examination of the LIPS, the BREATH, the SALIVA, the TEETH and the GUMS. We will consider the symptoms referable to these structures in that order.

§ 141. **The Lips.** *Dryness* of the lips is often one of the most conspicuous evidences of indigestion, and it is a very useful one, because this disorder has so few physical signs to assist us. The lips are *pale* in anæmia, they are *cyanosed* in advanced bronchitis with dilated right heart, and in other conditions where there is obstruction in the circulation. This cyanosis is especially marked in congenital heart disease.

FISSURES around the lips are an almost infallible sign of syphilis, especially when surrounded by a reddened infiltration. This infiltration helps us to distinguish a syphilitic fissure from the "cracked lip," which is the only condition liable to be mistaken for syphilis. *Cracked lip* occurs mostly in nervous children who lick and bite their lips, and are exposed to cold winds. It can generally be remedied by the application of some simple ointment,

such as zinc ointment or cold cream ; whereas the syphilitic fissures do not yield to this treatment. By pressing the corner of the mouth inwards and forwards, when the patient opens his mouth, we may often detect a mucous patch surrounding a syphilitic fissure inside the mouth. The *scars* left by syphilitic fissures are also a useful indication of a previous attack, or still more frequently that the patient has had congenital manifestations. They are white and stellate.

§ 142. **The Breath** should be normally quite free from any kind of odour. Offensiveness of the breath may arise from four sources. (1) A want of cleanliness *in the mouth*, particles of decomposing food, and the presence of decayed teeth, may give rise to a very offensive odour of the breath. (2) *Dyspepsia, constipation*, and other conditions of the *alimentary canal*, and the derangement of digestion in fevers, may also produce a bad smell of the breath. (3) Some *diseases of the nose* ; thus it always accompanies ozæna, which is a foul discharge from the nose. (4) A large cavity in the *lungs*, especially if *bronchiectasis* is present, produces a putrid odour. The foul odour of bronchiectasis is extremely powerful, and is characterised by being intermittent ; it comes on suddenly, lasts a day or two, and disappears gradually. Certain general conditions are attended by a more or less characteristic odour of the breath. Thus, in *diabetes* it is sweet ; in acute *alcoholism* it is alcoholic or ethereal. In *uræmia* it is said to be urinous. Certain *drugs* give rise to a very characteristic odour in the breath, *e.g.*, turpentine (a resinous odour), chloral (odour of chloroform), bismuth (odour of garlic), and opium (odour of the drug). Alcohol, ether, chloroform, and many other volatile substances are partly excreted by the breath.

§ 143. **The Saliva** may be *increased* (i.) in mouth inflammations ; (ii.) in chronic gastritis, in which there is such a profuse flow of saliva during the night that it gives rise to the impression that the patient, in the morning, is vomiting clear alkaline fluid (water-brash or pyrosis) ; (iii.) in pregnancy, mania, hydrophobia, and some other diseases ; (iv.) after the administration of mercury, pilocarpine, bitters, and according to some, alkalies and acids. The saliva is *decreased* (i.) in certain febrile states, (ii.) in diabetes, (iii.) severe diarrhœa, (iv.) chronic Bright's disease, and (v.) during

the administration of atropine or daturin. A condition known as “dry mouth” has been described by some authors,<sup>1</sup> in which there is a constant deficiency of saliva. The cause of this is obscure.

**The Palate** may be “cleft” from childhood, otherwise a hole in this situation is practically always an evidence of past syphilis. The *soft* palate shares in the diseases of the fauces (§ 111). It is a favourite position for the membrane of diphtheria, which in this situation forms an important means of differentiating the disease from follicular tonsillitis, the exudation of which never affects the palate. As regards the *hard* palate, it is sometimes involved in the diseases of the floor of the nose. A swelling may appear here in abscess of the antrum, or in abscess dependent on disease of the lateral incisor tooth. The latter is the commonest cause of swelling in this situation according to Tomes.<sup>2</sup>

*Thirst* accompanies all febrile conditions, and inflammatory conditions of the gastric mucous membrane. It is met with also in diabetes, after diarrhœa and vomiting, and after a diet excessively salted.

§ 144. **The Teeth** are subject to a certain amount of variation, even in health. The *average* dates of the eruption of the temporary and permanent teeth are as follows :—

<i>Temporary or “Milk” Teeth.</i>						<i>Permanent Teeth.</i>					
About 6th—8th month, lower central incisors.						About 6th year, first molars.					
About 8th—10th month, upper incisors.						“ 7 “ central incisors.					
About 12th—14th month, first molars.						“ 8 “ lateral incisors.					
About 18th—20th month, canines.						“ 9 “ anterior bicuspid.					
About 2—2½ years, posterior molars.						“ 10 “ posterior bicuspid.					
						“ 11—12th year, canines.					
						“ 12—13 “ second molars.					
						“ 17—25 “ third molars.					

One quarter of the mouth may be represented diagrammatically thus :—

Teeth ...	I.	I.	C.	M.	M.	Teeth ...	I.	I.	C.	B.	B.	M.	M.	M.
Month of eruption.	6 (or 7)	9	18	12	24	Year of eruption.	7	8	11	9	10	6	12	24

The normal order of eruption of the teeth may be represented

Clin. Soc. Trans., 1885.  
<sup>2</sup> “System of Dental Surgery,” Charles S. Tomes (London, 1897), p. 680.

thus :—MILK teeth 6, 9, 18, 12, 24 MONTHS ; and PERMANENT teeth 7, 8, (11), 9, 10 ; 6, 12, 24 YEARS. These details are worth remembering, because defective or deficient teeth are, in modern times, an extremely frequent cause of faulty digestion.

The presence of decayed teeth is one of the commonest causes of the dyspepsia of modern times, if not the commonest, and it is an ominous feature that a very large proportion of the candidates for the Army and Navy Services are rejected on account of bad teeth. They decay early in rickets, in cretinism, and in some other constitutional conditions.

The teeth are altered in shape after stomatitis in early life, which may be due to mercury, &c. (*vide* § 147). In these circumstances the teeth present transverse and vertical ridges, with or without alteration of shape. "Hutchinson's teeth" are alterations in the shape of the permanent teeth, due to hereditary syphilis, and present a valuable means for the identification of this disorder, as they are of very frequent occurrence in that disease, and bear lifelong testimony. The character of them is that they are both pegged and notched, that is to say, the transverse measurement is smaller at the free edge than the part near the gum, and on the edge of each tooth there are one or two notches (see Fig. 3, p. 25).

§ 144a. **Toothache** (odontalgia) is caused most frequently by decay (caries) of the teeth ; but there are other **causes**, the chief of which, as given by Mr. C. S. Tomes (*loc. cit.*), are as follows :—

1. Morbid conditions of the *tooth-pulp*—including irritation, acute and chronic inflammation of the pulp, pressure from confined matter in the pulp cavity, and deposit of secondary dentine in its substance.

2. Exposure of sensitive dentine, with or without caries, is probably the commonest cause of toothache—a "hollow tooth" as it is called.

3. Morbid conditions of the *alveolar periosteum* and exostosis—including inflammation of the periosteum, acute and chronic alveolar abscess, lesions manifested by alterations of the roots of the tooth (such as roughening by absorption or increase by exostosis).

4. Morbid conditions of the *periosteum of the jaws*—*e.g.*, traumatic, rheumatic, strumous, or syphilitic periostitis.

5. Irritation of the *dental nerves* by causes not productive of visible local lesions. This may include malposition of wisdom teeth, retarded eruption of wisdom teeth, and pressure due to insufficient space for the teeth.

6. *Inflammations and Ulcerations of the mucous membrane* and sub-mucous tissue—*e.g.*, that consequent upon difficult eruption of wisdom teeth and other conditions. See causes of stomatitis (*below*).



This is merely an approximate classification. The character and degree of the pain is greatly modified by the condition of the patient. The pain is generally more or less of an intermittent type. It is often absent at periods of full vigour—*e.g.*, after breakfast or dinner. Pain due to irritation, or to chronic local inflammation of the pulp, partakes of a neuralgic character, and the patient is often unable to point out the affected tooth or teeth. The suffering induced by acute inflammation of the pulp is excessive, particularly if there be no exit. It ceases more or less abruptly from the consequent death of the pulp. The recumbent posture or active exercise serves to aggravate the pain by increasing the vascular supply.

The **Treatment** belongs to the dental surgeon, but a good deal of temporary relief may sometimes be obtained by constantly rinsing the mouth with hot carbolic lotion, 1 in 100. A formula for drops to apply to a hollow tooth is given in F. 23 at end of book.

§ 145. **The Gums.** The pallor of anæmia, the purple line of lead poisoning, the red and ulcerated condition in stomatitis, the sponginess in mercurialism and scurvy are all useful local indications of some general condition. A swelling of the gums with greenish discharge suggests Actinomycosis (*q.v.*). *Bleeding from the gums* is apt to occur in scurvy, purpura, the hæmorrhagic diathesis, and even in apparent health, when the teeth are covered with tartar and the gums recede. In some people the gums very readily bleed, and sucking them may produce bleeding, which enables malingerers and hysterical persons to simulate disease of the lungs or stomach. It is detected by being only in small or moderate quantity, and by its intimate mixture with saliva. There is one disease of the gums, pyorrhœa alveolaris, the importance of which has only recently been recognised.

§ 146. **Pyorrhœa Alveolaris** (Riggs' Disease or Suppurative Gingivitis) is a raw ulcerating condition of the gums around the sockets of the teeth or stumps. When tartar is allowed to collect upon the teeth it gradually pushes the gum back; and by degrees a pocket or fossa is formed around the neck of each tooth, and there is considerable sero-purulent and often blood-stained discharge from the pockets thus formed, which not only imparts an offensive odour to the breath, but, being continually swallowed, is absorbed, and sets up a chronic toxæmic condition, which it is now recognised may produce a large number of troublesome symptoms.<sup>1</sup> Dyspepsia, even apart from difficulties of mastication, invariably ensues sooner

<sup>1</sup> This subject was the topic of an interesting discussion at Roy. Med. Chir. Soc., June, 1900. The blood-stained discharge is liable to be mistaken for serious disease of the stomach or lungs, as in cases mentioned at that discussion.

or later. But even before the dyspepsia becomes established the patient is listless, languid, and unfit for work, and complains of a great variety of functional nerve symptoms. A large proportion of my out-patients at the Nerve Hospital who complain of functional neuroses owe their troubles to pyorrhœa alveolaris. Among the symptoms due to this cause I may mention—headache, neuralgia, pains or tingling in the limbs and prostration, attacks of flushing, shivering or giddiness; a feeling of heaviness, and swelling of the limbs which is sometimes attended by actual œdema of the ankles, wrists, and other parts, which differs from ordinary anasarca in requiring longer pressure to produce the pit. Great depression is usual, and even melancholia may result; one of my patients committed suicide.

*Treatment.* All of these symptoms may arise when, for instance, stumps are left beneath an artificial plate. If the pyorrhœa, as frequently happens, be not identified as the cause and removed, no treatment is of much use. The tartar must be removed and the suppurating pockets carefully dressed with sulphate of copper or some other mild escharotic. This must be done at intervals, at first, of a week or so, later on a few months, by a skilled person, for the patient cannot do it himself. The only radical cure is the removal of the teeth or stumps.

§ 147. **Stomatitis** is a generalised inflammation of the mouth,<sup>1</sup> and it is evidenced by redness, swelling, tenderness and pain, of the mucous membrane, swelling and protrusion of the lips in severe cases, offensive odour of the breath; and usually, not always, excess of saliva. This, the simplest form of stomatitis, such as occurs in dentition or the application of caustics, is known as (a) *Catarrhal* or *Erythematous Stomatitis*. (b) *Aphthous Stomatitis*, also known as *Vesicular* or *Herpetic Stomatitis*, occurs in badly fed children, and it presents in addition to the above features, small grey patches, with a red base and sharply defined circular margin, resembling vesicles, which are very painful to the touch. (c) *Ulcerative Stomatitis* occurs in a mild and also in a severe form. In this we find, in addition to the features belonging to variety (a), irregular ulcers, especially on the gums, which recede from the teeth, so that the teeth become loosened. In the severe form there is great fœtor of the breath, considerable enlargement of the glands, submaxillary and cervical, and constitutional disturbance; and the teeth may drop out of the ulcerating gums. The ulcers often have a yellowish or grey coating, resembling a membrane, and it is therefore sometimes called *pseudo-membranous stomatitis*, or *phagedenic gingivitis*. (d) *Gangrenous Stomatitis*

<sup>1</sup> When the inflammation is confined to the gums the disease is known as *gingivitis*. *Pyorrhœa alveolaris* is described separately above.

(Cancrum Oris, Phagedena Oris, Noma Oris) is a gangrenous inflammation starting at one spot, usually on the cheek or on the lips. At first there is acute pain, but as this passes off a black spot forms (usually both internally and externally), which spreads peripherally, and leads to perforation of the cheek. The inflammation may spread to the gums, and the teeth become loosened. This is a severe disease, attended by considerable prostration, and, at first, a subnormal temperature. It is apt to follow measles or other exhausting illnesses in weakly children exposed to bad hygienic conditions.<sup>1</sup> (e) *Pyorrhœa Alveolaris* has been separately described above (§ 146).

*Etiology of Stomatitis.* (1) Certain *local conditions*, of which the commonest are dentition, tartar, and a want of cleanliness, the local irritation of a jagged tooth, excessive smoking, dirty feeding-teats in children, the application of hot fluids and caustics, new-growths simple or malignant, and gummata. In most of these cases the stomatitis takes the form of (a) or (b) above. Mouth-breathing and chronic gastric catarrh are said also to give rise to stomatitis occasionally, and necrosis of the jaw may lead to an Ulcerative Stomatitis.

(2) Certain *drugs and chemical substances* are apt to be attended by stomatitis. Chief amongst these is mercury, which gives rise to a very characteristic ulcerative stomatitis, with spongy gums and great fœtor of the breath. Arsenic and iodides may give rise to catarrhal stomatitis. Phosphorus produces ulcerative stomatitis, with necrosis of the jaw. The blue line of lead may be attended by a certain amount of catarrhal stomatitis.

(3) Chief among the *constitutional conditions* which cause stomatitis is (i.) the lowered vitality met with in phthisis and other wasting disorders; or in badly fed children, in whom the stomatitis may be aphthous or ulcerative. Thrush (Fig. 61) often accompanies catarrhal stomatitis in these circumstances. (ii.) Syphilis is accompanied by a special variety of the catarrhal form, and is attended by whitish, semi-transparent patches on the tongue and mucous membrane resembling "snail-tracks." Later on, ulcerations may occur (*vide* § 116, Throat). (iii.) Measles and other acute specific fevers are apt to be followed by cancrum oris in children exposed to bad hygienic conditions. Diphtheria is attended by both stomatitis and rhinitis when the membrane affects the mouth and nose. (iv.) Scurvy and purpura are attended by swollen and spongy gums and ulcerative stomatitis. (v.) Gastro-intestinal derangement, as in dyspepsia and fevers, leads not infrequently to catarrhal and sometimes aphthous stomatitis. (vi.) A lowered state of health, with insanitary environment, gives rise to epidemics of ulcerative stomatitis, sometimes taking a fatal form, in jails, hospitals, and camps. Occasionally this condition

<sup>1</sup> Microbes probably play a more important part than is at present recognised in the pathology of stomatitis, and the entry of these organisms into the mouth, where they may perhaps lodge on some chance abrasion, might possibly account for some of those hitherto inexplicable cases of the disease. For instance, it seems highly probable that that extremely serious condition, *Cancrum Oris*, may be connected with one of the group of bacteria which has lately attracted attention, and which includes the *Bacillus aerogenes capsulatus*, *B. œdematis maligni*, *B. emphysematosis*, and other microbes found in connection with "acute spreading traumatic gangrene," malignant œdema, *gangrene foudroyante*, &c. See, e.g., a paper by Corner and Singer on 'Acute Emphysematous Gangrene, the *Lancet*, November 17th, 1900; and Discussion at the Path. Soc. Lond. in the *Lancet*, 1900, vol. ii., p. 1651.

is met with in individuals in private life. Dr. Sibley<sup>1</sup> has published cases of ulcers in the mouth of neurotic origin (*Stomatitis Neurotica Chronica*).

(4) Certain *skin lesions* may invade the mucous membrane of the mouth, such as small-pox, chicken-pox, measles, and herpes iris. The eruption of measles is said to appear sometimes in the mouth before appearing on the skin, a fact which, if established, would aid in the early diagnosis of the affection.<sup>2</sup>

*Lichen ruber planus* may affect the mucous membrane of the mouth and tongue, and it may be present there even before it appears on the integument. In this situation it has a whitish appearance much resembling secondary syphilis, for which it has sometimes been mistaken. The distinguishing features are (i.) the smaller size and the circular or polygonal outline of the spots; (ii.) the evidences of syphilis or of lichen ruber planus respectively on other parts of the body.

*Prognosis of Stomatitis.* As a rule, stomatitis is not a serious disease, excepting that form known as phagedenic stomatitis, in which the mortality is 80 per cent. The catarrhal, aphthous, and ulcerative stomatitis generally end in recovery in a week or two. As regards the causation, those cases due to constitutional conditions are, as a rule, far more serious and obstinate than those due to local, or removable, conditions. The stomatitis of mercury may be extremely severe, but is, happily, only rarely seen nowadays. When aphthous stomatitis occurs in adults, accompanying a lingering disease, it is very obstinate, and is, in itself, a very grave omen. The prognosis is grave in the epidemic form, which is probably of microbic origin. The complications of the phagedenic form are diarrhœa, broncho-pneumonia, and gangrene in other parts of the body, especially the organs of generation (*noma pudendi*).

*Treatment.* In all varieties. The indications are, first, to remove the cause; secondly, to alleviate the local inflammation; and, thirdly, to attend to the general health. Any tartar present should be removed, and a wash for the mouth used, consisting of carbolic (1 in 100); or mercuric chloride (one grain to 6 ozs.); sod. bicarb. (1 in 20); or chlorate of potash or boracic acid (1 in 30). Chlorate of potash may be also administered with iron internally. In all pronounced cases milk and slop diet is indicated on account of the difficulty and pain of mastication.

*Aphthous and Ulcerative Stomatitis* are best treated by touching the sore places with solid nitrate of silver or sulphate of copper. For the pain, a solution of cocaine ( $\frac{1}{2}$  to 3 per cent.) may be used. In the ulcerative form chlorate of potash is especially useful. Antiseptics should be applied, *e.g.*, swabbing or painting the throat with glycerine and mercuric chloride (1 in 3), and a mouth-wash of 1 per cent. pot. permanganate. In the *Gangrenous* form (*Cancrum Oris*) prompt measures are necessary to avert a fatal issue. The modern treatment of this is averse to the application of strong caustics, such as were formerly used. The superficial sloughs should be gently removed by swabs, forceps and scissors, and the parts beneath swabbed with perchloride (1 in 1000) or carbolic (1 in 100), and this procedure should be repeated every hour, night and day, when the patient is awake. Abundance of stimulants and nourishment are called for.

<sup>1</sup> *Brit. Med. Journ.*, April 15th, 1899.

<sup>2</sup> *Koplik, Med. Record*, New York, April 1898.



### The Tongue.

The alterations to which the tongue is liable will be referred to under six headings: (a) Furring of its surface; (b) Ulceration; (c) White patches; (d) Acute swelling; (e) Chronic swelling (Hypertrophy), and Atrophy; (f) Warts, fissures, and cicatrices. A mother sometimes speaks of her child being "*tongue-tied*" when the frenum is too short. In some cases this is really so, or the structure may be attached to the tongue too far forward; but it exists much less frequently than parents suppose.

§ 148. **Furring of the tongue.** The appearances of the dorsum of the tongue used to be looked upon by older authors as indicating the state of the stomach; and with certain reservations it is still regarded as some aid in the investigation of that organ (§ 165), though it is a better guide in the prognosis of fevers and other grave constitutional disorders. Five varieties of tongue have been described by authors:—(1) The *pale, large, flabby tongue*, with broad tip and indented edges, and a uniform thin white coating, is the commonest abnormality. It is met with after alcoholic excesses, in atonic dyspepsia, in anæmia, in gouty persons, and in those the subject of lithæmia. (2) A *red tongue*, with sharp red tip and edges, in which the hyperæmic papillæ contrast strongly with the slight white coating in the centre, is found in subacute gastritis and irritable dyspepsia. (3) The *coated tongue*, with a uniform white layer over the surface, is found in acute gastritis, feverish conditions, anæmia, and nervous depression. Two forms of this tongue have been described: (i.) the *strawberry tongue*, having a slight white coating through which the fungiform papillæ protrude at the tip and edges, is very typical of scarlatina and other highly febrile states. (ii.) The *plastered tongue*, where the coating is considerably thicker. The amount of coating on a tongue varies directly with (a) the amount of dryness of the mouth, that is to say, the deficiency of salivary secretion (*e.g.*, in fevers and profuse perspiration); and (b) with the immobility of the tongue, owing to eating food that does not require mastication. The plastered tongue may pass on to—(4) the *furred tongue*. The coated papillæ stand out separately, giving a shaggy appearance. It is met with in states of marked prostration; *e.g.*, coma, abdominal cancer, advanced phthisis, profound anæmia, and other asthenic states. The prognosis is grave when the tongue becomes encrusted, and its

dryness increases. From any cause it may become dry, brown, and crusted, and then pass on to—(5) the *denuded red tongue*, which generally follows the preceding as the crust falls off. This tongue is red, shiny, smooth, and often cracked. It is found in advanced states of the preceding conditions, in diabetes, and other severe chronic ailments. The appearance of this tongue in a disease is of very grave prognosis. Aphthous stomatitis may supervene.

There is no doubt that large individual peculiarities in the character of the tongue exist apart from disease. On this account, some<sup>1</sup> go so far as to say that the tongue is of little importance as a clinical indication. Undoubtedly, we should make sure in any given case, that the tongue condition before us is not due to these personal peculiarities, to smoking in excess, or to previous disease.

As regards *treatment*, it is an old saying that a red tongue requires alkalis, and a white tongue acids. The former of these is true to some extent, but not the latter. With the exception of diabetes, a dry tongue indicates no appetite, and deficient gastric secretion; therefore the patient should be fed on fluids, animal soups, and other things requiring no great digestive power. (4) and (5) call for alcohol and other stimulants. In the prognosis of enteric fever the tongue is a valuable indication.

**§ 149. Ulcers of the tongue** may be Simple, Syphilitic, Malignant, or Tubercular.

I. SIMPLE ULCERS of the tongue are known by their superficial character, by the presence of some local cause, such as a jagged tooth or other local irritation (see also Ulcerative Stomatitis). The *frenum* is apt to be ulcerated in whooping-cough. This is probably of mechanical origin, but it is a useful aid in diagnosis.

II. SYPHILITIC ULCERS are of two kinds—(a) superficial, (b) deep.

a. *Superficial syphilitic ulcers* of the tongue are met with usually at the side, or in the form of fissures on the dorsum (see Fauces for description); or superficial circular “punched-out” ulcers.

b. *Deep syphilitic ulcers* are preceded by the formation of a roundish nodule (a gumma) which ulcerates; they are recognised by (i.) their site, which is usually on the centre of the dorsum; (ii.) their raised, ragged, and sometimes undermined edges; (iii.) the yellow slough which covers the base, and (iv.) the fact

<sup>1</sup> E.g., Mr. Jonathan Hutchinson, *Med. Press and Cir.*, July, 1883.

that they leave deep stellate scars. Syphilitic ulcers are usually multiple; the chief difficulty arises in the case of a single ulcer, as to whether it be syphilitic or cancerous. Syphilitic ulceration is differentiated by:—(1) the relative absence of surrounding induration, and consequently there is less interference with the movements of the tongue; (2) the site of the ulcer, on the dorsum; (3) there is less glandular enlargement, and the glands have a shotty feel; (4) the age of the patient, malignant ulcers rarely occurring before 40; and (5) there is a history of syphilis, and the disease *heals under iodide of potassium*.

III. MALIGNANT ULCER of the tongue is known by (i.) its site, which is chiefly on the side of the tongue; (ii.) its hard, raised, everted edges, and its uneven warty base, with foul discharge and tendency to hæmorrhage; (iii.) the induration around, and the early involvement of the glands; and (iv.) the early impairment of the movements of the tongue, with great pain.<sup>1</sup>

IV. TUBERCULAR ULCERS are not common. They are superficial with a yellowish discharge, and there is generally a history of tubercle in the lung or throat. The tubercle bacillus may be found in the scrapings.

*Prognosis.* Simple ulcers are easily dealt with, but other ulcers of the tongue are chiefly dangerous from their liability to hæmorrhage, and because of the important structures around. The diagnosis of syphilitic from malignant lesions is as important as it is difficult, for however advanced the former may be, they yield to appropriate remedies, but the latter are necessarily fatal, being certain to recur sooner or later after removal.

*The treatment* consists of the usual surgical measures. In syphilitic ulcers, iodide should be given in large doses. It is rarely given in sufficient quantity.

§ 150. **White patches** are not infrequently met with on the tongue, and may result from:—(I.) thrush; (II.) leucoplakia; (III.) aphthous stomatitis (§ 147); (IV.) syphilitic patches (§ 116). The two last are described elsewhere. The stellate cicatrices so characteristic of syphilitic lesions must not be confused with any of these.

I. In THRUSH (parasitic stomatitis) there are white membranous patches, like milk curd, sometimes with an arcola round them. They are

<sup>1</sup> These are the characters in an advanced case when diagnosis from syphilis is relatively easy. In an early stage it may be very difficult. In that stage a cancerous ulcer has flat sloping edges and scanty secretion, *its progress is very slow*, and it does not yield to iodides.

distinguished from other similar affections by (i.) leaving a bright bleeding surface when they are scraped off, and (ii.) on microscopic examination finding the fungus, *oidium albicans* (*saccharomyces albicans*, Fig. 61). It usually starts on the tongue, but may invade the lips and the whole of the interior of the mouth. The disease occurs chiefly in infancy, also in the later stages of exhausting diseases in adults. In the adult it only occurs at the end of wasting disorders, and not infrequently forms one of the indications of approaching dissolution. In infancy it generally arises in hand-fed children under bad hygienic conditions, and is often attended by diarrhœa. It is contagious from child to child. In children it has no very great significance, and readily yields to glycerine and borax, or weak carbolic lotion (1 in 500). The diet and methods of feeding should always receive attention in such cases. The patches may be removed by cotton wool swabs soaked in weak carbolic. In such children it sometimes happens that excoriations are noticed around the anus, and the mother thinks the "thrush has gone through the child"; but these are more frequently due to congenital syphilis or eczema intertrigo.



Fig. 61.—*OIDIUM ALBICANS*, OR  
THRUSH FUNGUS.

II. *LEUKOPLAKIA LINGUÆ* (syn. *Ichthyosis Linguae*) is a term applied to flat whitish horny-looking silvery patches on the tongue; due to a heaping up and condensation of the epithelium. The disease generally involves a considerable area of the tongue. In a later stage the tongue becomes red and glazed. The patches themselves are often cracked, and form a pavement-like surface, which has the appearance of ichthyosis of the skin. They give rise to a great deal of discomfort and tenderness. It is most frequently met with in tertiary syphilis, and, according to most observers, it is always an evidence of that disorder. But others attribute the condition to excessive smoking, jagged teeth, drinking, and dyspepsia. I cannot say that I have met with a case which

could not be attributed to syphilis. *The treatment* is, as a rule, very unsatisfactory, unless the disease be met with in the early stages. A mouth-wash, consisting of bicarbonate of soda (20 grains to the ounce), or a saturated solution of chlorate of potash, sometimes relieves the symptoms. But the best treatment, in my experience, is the local application of chromic acid (5 to 10 grains to the ounce, gradually increased) painted on daily. It should be accompanied by antisyphilitic remedies, though they do not have a very marked effect. Some say that strong or irritating applications should be avoided.

§ 151. **Acute swelling of the tongue**—*i.e.*, swelling of the tongue coming on rapidly—may be due to either (a) *acute glossitis* or (b) *acute œdema*. In both of these the tongue rapidly enlarges, and may even protrude beyond the teeth; a great deal of pain is present, and there is a difficulty of swallowing and speaking.

a. **ACUTE GLOSSITIS** may be due to various local causes—*e.g.*, the sting of an insect, a septic wound, biting of the tongue, acute ulcers—or, it



may be due to constitutional conditions—*e.g.*, mercurial salivation, and, according to some, acute specific diseases, such as erysipelas. The onset of acute glossitis is rapid, though rather less so than the œdema about to be described, the swelling rarely extends beyond the tongue, and the glands are more markedly involved. Its *treatment* consists of the use of mouth-washes, and especially chlorate of potash, the painting of a cocaine solution (10 per cent.), the administration of chlorate of potash, iron, and bark internally, with purging and antiphlogistic remedies generally. Free incisions may be necessary.

b. ACUTE ŒDEMA of the tongue is a serious disorder because of its liability to involve the glottis. It may accompany urticaria, or it may be, like the angina of Ludovici (§ 120), of an erysipeloid nature. The œdema comes on suddenly, and in the course of a few hours the tongue may protrude from the mouth. The swelling rapidly extends to the throat, nose, and down the œsophagus and trachea. It is attended by an inability to speak, to swallow, and sometimes even to breathe. Its *causation* is somewhat obscure, but it is said to be usually of an urticarial nature, and to occur in those who have had urticarial attacks, in which case it may come on after certain articles of food, *e.g.*, shell-fish. This condition is *diagnosed* from simple acute glossitis by (i.) its rapid advent in the course of an hour or two; (ii.) the rapid extension to the throat and other parts; (iii.) the presence sometimes of an urticarial rash upon the skin. Without the last feature the diagnosis is somewhat difficult.

*Prognosis and Treatment.* The disease comes on rapidly, and runs a very rapid course, subsiding in the course of twenty-four hours, unless the patient die in the meantime. It is apt to cause suffocation. Prompt measures are necessary. A strong purge should be given at once (croton oil 1 ℥ if it can be swallowed) or a turpentine enema. Cocaine (5 or 10 per cent.) should be kept constantly painted on the tongue. Scarifications and leeches may be required, and the practitioner should be prepared to perform tracheotomy if necessary.

§ 152. **Hypertrophy and Atrophy of the tongue.** HYPERTROPHY, OR CHRONIC SWELLING OF THE TONGUE, may be due to chronic glossitis or macroglossia.

I. *Chronic glossitis* is a chronic inflammation of the tongue, in which either the surface or the substance is mainly involved. The *surface* is covered with irregular, red, raw, tender patches and cracks (unless it be secondary to, or attended by, leucoplakia). If the *substance* be affected, the organ is enlarged, indented by the teeth, and in course of time it becomes indurated. It is more frequently due to some local irritation, such as a jagged tooth or an ulcer, in which case the enlargement is generally limited to one part of the tongue. Glossitis may arise from alcoholism, chronic dyspepsia, or excessive smoking; and many of the other causes of stomatitis (*q. v.*). The treatment is directed to the removal of the cause, and the employment of chlorate of potash and astringent mouth-washes.

II. *Macroglossia* is due generally to a congenital overgrowth of the connective tissue, accompanied by a dilatation of the lymphatics of the tongue. Its causes are obscure. Persistent application of mild caustics, or the galvanic cautery to the tongue is the only remedy.

ATROPHY OF THE TONGUE (microglossia). Atrophy of the tongue may arise from nerve lesions. It may occur in bulbar paralysis, and is

then usually bilateral. In unilateral cases the lesion is either situated in the nucleus or trunk of the twelfth nerve of one side (*vide* Chapter XIX.).

### § 153. Warts, Fissures, and Cicatrices.

WARTS are simple or syphilitic. *Simple* warts are distinguished by the fact that they are soft; they are raised and often pedunculated, and there is but little secretion. The glands are not shotty to the touch. *Syphilitic* warts are hard, with infiltration; they are never pedunculated, secretion is present, and the glands of the neck and elsewhere are shotty.

FISSURES are also divided into simple and syphilitic. The *simple* can generally be accounted for by some such cause as the irritation of a ragged tooth, and are never infiltrated. On pinching *syphilitic* fissures between the fingers, infiltration is found to be present.

CICATRICES. Simple ulceration rarely leaves a scar, but if so it is never hard. Hard stellate scars are invariably indicative of syphilis.

## The Gullet.

§ 154. SYMPTOMATOLOGY.—Diseases of the œsophagus have practically one symptom which is common to all, namely, *dysphagia*, *i.e.*, a difficulty in swallowing. There are certain features about this symptom which it is important to investigate:—

*First*, does the difficulty apply to both liquids and solids? This gives us an idea of the *degree* of the obstruction. *Secondly*, does the food return; and if so, after what interval? This is sometimes a guide to the *seat* of the obstruction. Obstruction within the *œsophagus* has to be distinguished from obstruction at the pyloric end of the *stomach*—(i.) by the easy way in which the food regurgitates as compared with the vomiting which accompanies pyloric stricture; and (ii.) by the absence of acidity in the matters returned. *Thirdly*, is there any pain? What is its situation, and is it only present after the ingestion of food? Constant pain is a feature of malignant disease. *Fourthly*, what is the duration of the dysphagia? Has it been persistent, and become progressively and steadily worse? The last-named is the leading feature of organic, as distinguished from functional dysphagia, which is frequently intermittent and by no means progressive. *Fifthly*, is there any regurgitation through the nose? This feature implies paralytic dysphagia with paralysis of the soft palate. *Sixthly*, is there any emaciation, or are there any symptoms referable to other organs? Marked emaciation coming on early in a patient beyond middle life is characteristic of carcinoma.

§ 155. *PHYSICAL EXAMINATION.*—(a) A careful *inspection* of the throat should be made, because the dysphagia may arise from tonsillitis or other pharyngeal conditions. The paralysis of the palate which succeeds diphtheria may thus be detected. Any swelling should be carefully examined, such as retro-pharyngeal abscess or tumour, or a foreign body in this situation. I have known the bristle of a tooth-brush in the pharynx give rise to very serious difficulty in swallowing.

(b) In cases of dysphagia of any duration *the passage of an œsophageal bougie*, or at any rate a soft stomach-tube, should always be made.<sup>1</sup> The solid bougie is preferable, both for purposes of diagnosis and of treatment; but if carcinoma be suspected, great care must be exercised. The chest should always first be examined for aneurysm, and if this be found the bougie should be avoided. The bougie must first be dipped in hot water in order to make it more flexible, and glycerin if necessary for lubrication. There is not much fear of it entering the larynx, provided the tube be passed to one or other side, and instruction given to the patient to put his head horizontally forwards and swallow during the operation. As the entrance to the stomach—from the teeth to the cardiac orifice—is a distance approximately of 16 inches, it is a good plan to tie a thread round the bougie 16 inches from its point, then one can tell when it has reached the stomach. The œsophagus starts at the cricoid cartilage, opposite the 6th cervical vertebræ, and ends opposite a point between the 9th and 10th dorsal vertebræ, a distance of 10 inches. The presence of acute *pain* during the passage of the instrument indicates ulceration, either simple or malignant. The presence of *blood*, and perhaps cancer cells adhering to the end of the tube, should be looked for as having the same significance as the foregoing. The presence of *dilatation* may be suspected when the end of the tube is not gripped, but is loose and easily movable. Occasionally a diverticulum or saccule of the œsophagus is formed which by its pressure on the gullet above or below it causes obstruction. In such cases a bougie which could not be passed before, may be passed after vomiting has occurred.

<sup>1</sup> Dr. J. S. Bristowe, with characteristic candour, narrates a case showing the consequences which arose from a neglect of this procedure, in his "Clinical Lectures and Essays," p. 43. The case was really one of dilatation of the œsophagus, which remained undiscovered until after death.

(c) *Auscultation* affords a valuable means of detecting both the presence and position of an œsophageal stricture. Place the chest end of a binaural stethoscope over the interval between the xiphoid cartilage and the left costal arch. Two gurgling sounds can be heard in this situation if the patient swallows *one* gulp of fluid; the first is when it passes from pharynx to œsophagus, the second is when it passes from œsophagus to stomach. The normal interval between these two is *six seconds*, but if there be any obstruction in the gullet this interval becomes increased. If the first sound cannot be distinctly heard, the moment of its occurrence can be judged by looking at the throat. Again, by placing the stethoscope on the left side of the neck in a healthy person a gurgling sound will be heard during the act of swallowing. This normal sound may be *traced round and down the back* on the left side of the vertebral spines as low as the 10th D.V. But if a stricture be present it will be delayed or *absent below the seat of stricture*.

§ 156. **Causes of dysphagia.** “When a patient complains of difficulty in swallowing, or that the food returns to his mouth, the practitioner should first think of thoracic aneurysm, secondly of cancer, and thirdly of some other kind of ulceration.”<sup>1</sup> The COMMONER CAUSES are :—

- I. A tumour pressing upon the gullet from the outside.
- II. Epithelioma (cancer) of the gullet.
- III. Simple or non-malignant stricture.
- IV. Spasm.
- V. Foreign bodies, acute œsophagitis, and simple ulcer.

LESS FREQUENT CAUSES are :—

- VI. Paralysis of the gullet.
- VII. Dilatation of the gullet.

§ 157. **A tumour** pressing upon the gullet from without is perhaps the commonest cause of dysphagia, although malignant or simple stricture and muscular spasm are regarded by many as of equal frequency. Any intrathoracic tumour may, by its pressure, narrow the lumen of the gullet, and undoubtedly the commonest of these is aneurysm of the aorta. Other tumours are—cancer of a neighbouring viscus, retropharyngeal abscess or tumour, enlargement of the bronchial glands, lympho-sarcoma or other mediastinal tumour, goitre, pericardial effusion, and diverticula of the gullet

<sup>1</sup> Bryant, quoted by Fagge and Pye-Smith, “Prin. and Pract. of Med.” vol. ii., p. 316 (2nd ed.)



filled with food (§ 163). The features common to all such tumours are the slowly progressive character of the dysphagia, the symptoms of pressure on other viscera, and sometimes, although usually not until late in the case, the physical signs of the tumour in question. For the rest, the differential features vary according to the nature and position of the tumour. In *aortic aneurysm* the amount of dysphagia is rarely very great at any time, although it is slowly progressive. Rest in bed will generally ameliorate the dysphagia. Difficulty of swallowing is only one of the pressure symptoms in this disease, and others should be looked for, *e.g.*, dyspnœa, abductor paralysis of the left vocal cord, and inequality of the pupils. The physical signs of aneurysm are commonly wanting in such cases, on account of its deep-seated position.

§ 158. **Malignant disease** of the œsophagus is due in the large majority of cases to an epitheliomatous growth in the wall, usually primary, which goes on to ulceration, and forms a stricture from 1 to 4 inches long. Rarely, the deposit is sarcomatous. The diagnostic features of epithelioma of the œsophagus are:—  
i. The patient is past middle life. It is said to be more common in males. ii. The dysphagia becomes steadily and progressively worse. At first a difficulty only exists with solids, but later on fluids also are returned. The duration of the whole illness rarely exceeds twelve to eighteen months. iii. Emaciation and other evidences of cachexia occur quite early in the illness. There may be evidences of cancerous deposit elsewhere, especially within the abdominal cavity. iv. Pain and hæmorrhage, those frequent accompaniments of all malignant growths, are usually present, and the pain is persistent and independent of, although aggravated by, food. v. The passage of a bougie is attended by considerable difficulty. The favourite sites of malignant stricture are opposite the cricoid cartilage, 6 inches from the teeth, opposite the bronchi, 9 inches, and at the cardiac orifice, 16 inches from the teeth.

*Fibroma* and *Myoma* and other benign growths in the œsophagus, sessile or in the form of polypi, are very rare. They may simulate simple or malignant stricture, and there may be hæmorrhage: but the absence of any cachexia and the long duration without any increase of symptoms are the only means of suspecting the condition.

§ 159. **Simple or non-malignant stricture** of the œsophagus is most frequently caused either by the narrowing due to a syphilitic

deposit, or the contraction which it subsequently leaves. It may also arise from the cicatrisation which follows a simple ulcer of the gullet or stomach; or, thirdly, as the result of swallowing a corrosive liquid. Dilatation may take place above the stricture. The differential features of this condition are:—i. The dysphagia comes on gradually, and, having reached a certain degree, is apt to remain stationary; the patient may be unable to swallow solids, but lives for many years on liquid food. ii. The passage of bougies gradually increased in size is possible, and this procedure gives some relief. iii. The patient may be young or he may be of any age; the cachexia and emaciation of cancer are wanting; and pain is not a prominent feature in the case. iv. The gullet is apt to dilate above the stricture and the food returns after an interval which becomes progressively longer as the dilatation becomes greater. v. There is nearly always a history of one of the three causes above mentioned.

§ 160. **Spasm of the pharynx or œsophagus** is in the author's experience one of the commonest causes of dysphagia. It is not infrequently associated with hysteria and other functional neuroses. Its differential features are fairly characteristic:—i. The dysphagia is never progressive. It may come on somewhat suddenly, dating perhaps from an emotional shock or trouble; and it is very often intermittent, the patient being well enough in the intervals. Sometimes solids can be taken, while fluids are regurgitated, or *vice versâ*. ii. It is unaccompanied by emaciation or cachexia; indeed, the patient sometimes appears to be in perfect health, a feature in which it differs from all other causes of dysphagia. There is usually little or no pain; and never any bleeding. iii. The dysphagia may last intermittently for a considerable time. I have known cases persist in varying degree for seven, twelve, and sixteen months. iv. The passage of a bougie, or flexible stomach-tube, is possible with a little steady pressure; and with the patient under chloroform it is easily done. This procedure generally results in curing the condition, at any rate for a time. v. The patient is most frequently of the female sex, and often presents other evidences of the hysterical diathesis. It certainly occurs also in males, and the gouty or rheumatic diatheses are said to predispose to it.

**161. Foreign bodies, acute œsophagitis, and Simple Ulcer.** The symptoms of these conditions are much alike. Acute œsophagitis occurs after traumatism, as after swallowing corrosive fluids,<sup>1</sup> or in a localised form from the presence of foreign bodies. It sometimes occurs in the course of the specific fevers, and in infants at the breast from unknown causes. A slighter degree of *localised* inflammation arises by no means infrequently when a fish-bone, needle, pin, bristle of a tooth-brush, or other solid particle, sticks in the folds of the œsophagus. This dysphagia takes the form of a difficulty and pain during the act of swallowing, at one particular spot. The symptoms here start suddenly and reach a maximum at once. This source of trouble is very apt to be overlooked when the patient has forgotten the incident which led to the lodgment of the foreign body. When the inflammation is *generalised*, there is great pain, with consequent spasm and regurgitation on attempting to swallow. Thirst, and, if the condition be severe, feverishness are present. Mucus, pus, and blood may be vomited if ulceration ensue.

*Simple Ulcer* of the gullet is very rare. It is sometimes due to syphilis. Acute pain and tenderness are prominent features; with spasm on swallowing or attempting to pass bougie. But the affection cannot be diagnosed with certainty.

We now turn to the rarer causes of **Dysphagia**.

**§ 162. Paralysis of the gullet.** Paralysis of the upper part of the gullet, *i.e.* of the *pharyngeal constrictors*, is not uncommon as an accompaniment and complication of diphtheria. Difficulty of swallowing under these circumstances may be one of the first evidences of diphtheritic paralysis. It also occurs in Bulbar Paralysis; and it occurs at the end of some slowly progressive exhausting diseases. All of these instances differ from the other causes of dysphagia by being attended by regurgitation of fluids through the nose, owing to the paralysis of the soft palate. Paralysis of the gullet *below the pharynx* is a much rarer condition. It may sometimes accompany, and be due to the same causes as the above. It also arises as an occasional complication of general paralysis of the insane, cerebral tumour, diseases of the nuclei in the medulla, and lesions of the vagus. The dysphagia in these cases is not absolute, the normal œsophageal sound on auscultation is absent, and a *bougie passes* without hindrance. The condition can only be distinguished from simple dilatation when there is no regurgitation or pseudo-emesis of food.

**§ 163. Dilatation or diverticulum of the gullet** is not frequent, and the causes are obscure. It may take the form of (a) A *generalised*

<sup>1</sup> Fluids which are simply irritating, such as beer contaminated by the substances used to clean the pewter pots, may cause the condition.

*dilatation* of the whole tube. (b) A *fusiform dilatation* above a stricture. (c) It may occur as a *diverticulum* or sac, which is said to be formed in one of two ways:—(i.) a *pressure* diverticulum or sacculæ, due either to weakness of the wall after injury, or sometimes to congenital weakness, of some part of the tube, with consequent hernia of the mucous, through the muscular, wall; and (ii.) a *traction* diverticulum, due either to adhesions between the œsophagus and neighbouring glands, or other structures, pulling out the œsophageal wall as they contract.

(a) A general dilatation has but few or no symptoms. (b) The symptoms of variety (b) are masked by those of the stricture below. The occurrence of dilatation (with stricture) is evidenced by the regurgitation of food at shorter or longer intervals. On this account such cases are very apt to be mistaken for the vomiting of pyloric obstruction (*vide* feature 2, § 154). There is an unusual mobility in the bougie just before it reaches the obstruction. (c) The diverticular varieties are very rare; but as far as we know their symptoms are as follows:—(i.) There is regurgitation of food after an interval varying from a few minutes to a few hours after ingestion. It is apt to be mistaken for persistent vomiting, but the ease with which the food is returned, and the absence of acid in the matters ejected, should make us suspect the condition with which we have to deal. (ii.) The regurgitation gradually increases in amount, and the breath is foul from the decomposition of food in the gradually enlarging pouch. (iii.) In cases of pressure diverticula a bougie which could not be passed before, can be passed after, vomiting; because the sac full of food forms a swelling that presses upon the gullet, and so leads to obstruction.

§ 164. PROGNOSIS AND TREATMENT OF DYSPHAGIA. Dysphagia is in most cases a symptom of considerable gravity, and in severe cases it commonly enough results in death by starvation. Of all causes, malignant stricture is the most serious, and, in spite of the means which modern surgery has placed at our disposal, patients rarely live more than a year or eighteen months. The length of time depends on the maintenance of the nutrition of the individual. Next in order of gravity come tumours pressing on the œsophagus, when the prognosis depends on the nature of the tumour and its amenability to treatment.

Patients with simple stricture, and with dilatation, may live for many years on fluid diet, with or without gastrostomy; but diverticula are much more serious. Of all causes functional spasm is the most curable, although it is apt to return.

The cause of death in dysphagia is usually starvation, or a low form of pneumonia. This may arise from perforation into the bronchus, or by the food passing into the glottis; in either case death is expedited by the lowered vitality of the patient. Perforation may occur in other directions; *e.g.* a case of malignant



disease of the gullet under my care died from hæmorrhage consequent upon perforation into the aorta.

TREATMENT OF DYSPHAGIA. The indications are to remove the cause of the obstruction, to maintain the strength and nutrition of the patient, and to relieve any concurrent symptoms. The question of three surgical procedures may arise in these cases—the passage of bougies of different sizes, the use of Symonds' tubes, and gastrostomy. If possible a bougie should be passed in all cases not only for purposes of diagnosis, but also as part of the treatment. It may be of little use in malignant stricture, but simple stricture may be dilated or prevented from further contracture by this method. Symonds' tube, a funnel-shaped tube with a string attached to prevent it slipping down, changed every three weeks or so, undoubtedly prolongs life both in malignant and advanced simple strictures. In malignant stricture if, when the case comes under treatment, debility is very marked, complications are present, and there are evidences of cancer elsewhere, neither Symonds' tube nor gastrostomy are of any use, and the only treatment available is rectal feeding. In addition to the above treatment the only indication in *malignant* stricture is to soothe the pain by morphia, opium, or cocaine.

In *simple* stricture, bougies of gradually increasing size should be passed and left in for some hours at a time. Force must not be used in so doing. In very narrow strictures a Symonds' tube would be better. If syphilis be suspected as the cause, potassium iodide must be given. In *functional* spasm a bougie should be passed, and cold douches given along the neck and the spine. The general condition must be treated, valerian in hysteria, combined with special diet in cases with gastritis. Electricity may be useful. In *paralysis* and *dilatation*, especially the diverticular type of dilatation, the patient must be fed by a stomach-tube. If the diverticulum is high up in the neck, the surgeon may be able to remedy it. In *acute œsophagitis* the pain must be soothed by morphia hypodermically, by cocaine lozenges, or by opium given with tragacanth. Thirst may be allayed with spoonfuls of iced water, in which small doses of opium, cocaine and milk may be administered. During the acute stage the patient may require nutrient enemata. *Foreign bodies* in the

gullet need prompt attention but very careful measures, else they may pierce the tube and injure the aorta or other structures around.

*Feeding by a stomach-tube* is a measure available in a fair proportion of cases, especially in causes I., III., IV., VI., and VII. (*supra*). The only apparatus necessary consists of a long flexible rubber tube (5 ft. long), one end of which is blunt with the "eye" at the side or the end (according to choice), and the other end tied to a funnel. The method of passing the tube is either the same as that used in passing a bougie (§ 155), or it is passed through the nose. In the latter case the size must be smaller. The operator then stands upon a chair, and pours in the fluid food, previously prepared, from a jug.

## CHAPTER IX.

### THE ABDOMEN.

THE abdomen contains a large number of very important organs and structures, but just as their physiology and pathology are in many instances obscure, so also are the means at our disposal for their thorough clinical investigation imperfect. However, it is in this region that we have to deal with symptoms which on the one hand may be of quite a trivial order, or on the other may be of extreme gravity ; symptoms and conditions the issue of which will largely depend on the promptitude, knowledge and skill of the medical man in attendance, and upon his adequate comprehension of their true meaning.

#### *PART A. SYMPTOMATOLOGY.*

§ 165. **Local Symptoms.** The symptoms referable to disease situated within the abdominal cavity are necessarily of the widest and most varied kind, but there are only three which are sufficiently constant to be regarded as cardinal symptoms, all of which are local, viz., ABDOMINAL PAIN, GENERALISED ENLARGEMENT, and LOCALISED TUMOUR.

VOMITING is a fairly constant accompaniment of all acute abdominal conditions whether the stomach is involved in the lesion or not. Its causes are discussed in § 192.

The presence of DIARRHŒA and CONSTIPATION depends very largely on whether the intestinal canal is affected, and these are fully dealt with in Chapter XI. The other symptoms also depend largely upon which of the abdominal organs is affected, with one important exception, viz., "INDIGESTION." In all chronic abdominal disorders, no matter which organ is affected, we are often consulted for "INDIGESTION" ; and, in point of fact, nausea and all the other symptoms of pronounced dyspepsia may be due to disease quite unconnected with the stomach and located, for instance,

within the uterus, kidneys, liver, spleen, or pancreas. Many a case of "dyspepsia" after resisting treatment for months or years has been cured by the stitching up of a dislocated kidney.

ABDOMINAL PAIN, if acute and sudden, is a medical emergency of the most important kind; if chronic, it presents many difficult questions for diagnosis. It will therefore merit the most careful study and analysis (§ 169). The diseases *outside the abdomen* which may give rise to it are only three in number:—

1. *Diaphragmatic pleurisy*, or a basal pleuro-pneumonia, may give rise to acute abdominal pain of sudden onset; to abdominal rigidity, and other symptoms of acute peritonitis, which can only be differentiated by the pulse-respiration ratio and the concurrent symptoms.

2. *Neuralgia* of the intercostal and other spinal nerves may be referred to the abdomen. In this way spinal caries, especially in children, may be mistaken for various abdominal diseases.

3. An *abscess* within the abdominal wall may be similarly mistaken, but this ought to present no difficulty.

ABDOMINAL ENLARGEMENT and ABDOMINAL TUMOUR are fully considered in Part C.

§ 166. The **general or remote symptoms** met with in abdominal disorders are, as just mentioned, of an extremely varied nature, and our endeavour should be to correctly associate these symptoms with the abdominal organ which is affected.

**COLLAPSE and PULSE-TEMPERATURE RATIO.** In connection with the general symptoms of abdominal diseases one fact needs special mention, namely, the profound collapse which is so apt to be associated with all acute abdominal conditions. A blow on the abdomen may result in fatal collapse, and so also may perforative peritonitis. A person with endocarditis, pleurisy, or even pneumonia, may walk to our surgery, but with any acute abdominal condition he takes to bed instinctively. This tendency to collapse probably finds an explanation in the fact that the chief centre of the sympathetic is situated within the abdomen. Now, a subnormal temperature is one of the symptoms of collapse, and for this reason the temperature rarely ranges very high even in the gravest abdominal inflammations, especially in their earlier phases. In acute peritonitis, for instance, an extensive inflammatory process affects the peritoneum, which acting alone might produce a temperature



of  $105^{\circ}$  or more, but by reason of the collapse it is rarely more than  $102^{\circ}$  or  $103^{\circ}$ . In the pulse, however, we find our best guide to the severity of the mischief within the abdomen. In all acute diseases, other than abdominal, we find a rough general proportion between the height of the temperature and the rate of the pulse. Thus, a temperature of  $100^{\circ}$  F. will correspond roughly with a pulse of 100,  $101^{\circ}$  with 110,  $102^{\circ}$  with 120,  $103^{\circ}$  with 130, and so on—an increase of about 10 for every  $1^{\circ}$  F. But in acute abdominal conditions this is not so. The pulse-temperature ratio is disturbed, for although the pulse rate increases with the severity of the abdominal mischief, the temperature never increases proportionately. Indeed, in many of the worst cases, the temperature is one or more degrees below normal. The pulse, however, is an almost infallible guide, and one may say (1) that if the pulse remains under 100 nothing very serious is happening within the abdomen; and (2) that the rate of the pulse, and the pulse-temperature ratio, are great aids to the diagnosis, and in some sense measures, of acute abdominal mischief; especially when that mischief has reference to the peritoneum.

#### PART B. PHYSICAL EXAMINATION.

§ 167. In the examination of the abdomen we must proceed systematically, as in the examination of the thorax, by INSPECTION, PALPATION, PERCUSSION, MENSURATION, and occasionally auscultation; though of all these measures palpation by the educated hand is at the present time the most valuable means we have.

1. INSPECTION of the abdomen should on no account be omitted, because much can be learned in this way. The best point of view is that from the foot of the bed, or by bending over the patient's feet, so as to view the abdomen from below. The mere fact of enlargement may thus be verified, and whether the enlargement be a generalised and uniform one, or whether it be localised. Notice whether the umbilicus is centrally situated, and also whether the surface presents dilated veins such as occur in abdominal cancer, or when the portal vein or vena cava is obstructed.<sup>1</sup> Notice also whether there is any thickening or

<sup>1</sup> Dilatation of the abdominal veins is met with chiefly in 3 conditions: (1) In liver cirrhosis, these veins being part of the conservative collateral circulation which gradually becomes established; (2) the veins without being much dilated or prominent are *widely*

infiltration round the umbilicus such as occurs in cancer and tubercular peritonitis. An abdominal enlargement due to the presence of air or gas is rounded anteriorly, but when due to fluid it is usually flattened in front and the flanks bulge; when it is due to the presence of a solid tumour it is irregular. Incidentally you may notice the presence or absence of the white lines (*lineæ albicantes*) left by a previous pregnancy, the knowledge of which may be medically useful.

THE REGIONAL ANATOMY OF THE ABDOMEN is important as a guide to the seat of disease (Fig. 62).

2. PALPATION. Considerable experience is necessary for satisfactory abdominal palpation. The hand should be warmed and always laid *flat* on the abdominal wall; then by gently dipping in the fingers, by flexing the metacarpo-phalangeal joints, we have the most ready method of ascertaining (1) the presence of any tumour, (2) the boundaries of some of the solid organs. The patient should lie on his back with the knees drawn up and the shoulders somewhat raised, so as to relax the abdominal muscles. Do not use the tips but only the pads of the finger, for they stimulate the recti muscles to contract, and thus to simulate a tumour where none exists. Many patients offer considerable involuntary or voluntary resistance, and this must be overcome by placing them in an easy posture and distracting their attention, or asking them to "let the breath go." The palpation and percussion boundaries of the different organs are described in later chapters.

3. PERCUSSION of the abdomen is done with the same precautions as in the case of the heart and lungs; and the student will now find it very convenient to be able to percuss with either hand indifferently. Normally the anterior surface of the abdomen is resonant (when the stomach and intestines are empty) as far upwards as liver and spleen, downwards as far as the pubes, and outwards as far as the outer border of the colon. By this means

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*apparent* in cases of abdominal carcinoma. It is a sign of considerable value and constancy. (3) Extreme dilatation and varicosity of the superficial veins occurs only when the inferior vena cava is obstructed. This is generally due to a gummatous deposit in or around the posterior border of the liver where the vena cava passes through it. The veins of the legs and testes generally share to a less extent in the dilatation. A case is recorded by Dr. W. Chapman, Clin. Soc. Trans., 1899 and 1900, and *Lancet*, December 2nd, 1899,

we ascertain the presence of solid and fluid, which are dull ; or of gas, which is resonant.

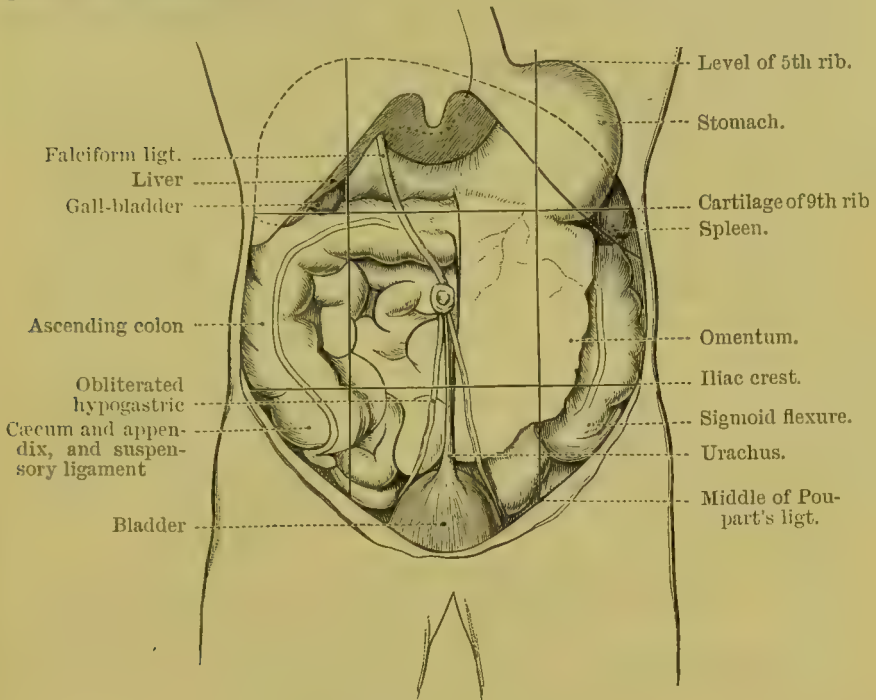


Fig. 62.—REGIONS OF THE ABDOMEN.

For purposes of convenience the abdomen is divided into nine regions, which are bounded by two imaginary lines running vertically upwards, on each side of the abdomen, from the *middle* of Poupart's ligament to the costo-chondral articulation above, and two horizontal lines running round the abdomen on a level with the end of the 9th costal cartilage, and the anterior superior spines respectively. Their names and the organs they contain are as follows :—

*Right Hypochondriac.*

The right lobe of the liver and the gall-bladder, the duodenum, pancreas, hepatic flexure of the colon, upper part of the right kidney, and the right suprarenal capsule.

*Right Lumbar.*

Ascending colon, lower part of the right kidney, and some convolutions of the small intestines.

*Right Iliac.*

The caecum, appendix caeci, and ureter.

*Epigastric Region.*

The middle and pyloric end of the stomach, left lobe and lobulus spigeli of the liver, and the pancreas.

*Umbilical Region.*

The transverse colon, part of the great omentum and mesentery, transverse part of the duodenum, and some convolutions, and the jejunum and ileum.

*Hypogastric Region.*

Convolutions of the small intestines and the bladder in children and in adults when distended, and the uterus during pregnancy.

*Left Hypochondriac.*

The splenic end of the stomach, the spleen and extremity of the pancreas, and the splenic flexure of the colon, upper half of the left kidney and the left suprarenal capsule.

*Left Lumbar.*

Descending colon, part of the omentum, lower part of the left kidney, and some convolutions of the small intestines.

*Left Iliac.*

Sigmoid flexure of the colon and ureter.

4. By MEASUREMENT we ascertain the amount of increase in size. As a general rule, horizontal measurement should be taken at the

level of the umbilicus; and it should be recorded for future reference. In order to ascertain whether the enlargement is symmetrical we measure from the umbilicus to the ensiform cartilage above and the pubes below, and from the umbilicus to the anterior superior spine on each side. These four measurements should be approximately equal. From these data we ascertain very slight deviations from symmetry.

The FALLACIES of abdominal enlargement are—1. *Fat in the omentum* is referred to under fluid enlargement (§ 186). 2. *Phantom tumour* is described under abdominal enlargement due to gas (§ 184). 3. *Pendulous abdomen*, so frequent in elderly women, is often thought by the patient to be a “tumour,” but it is due only to weakness of the muscles of the abdomen and of the intestinal tube. 4. *Pregnancy* is referred to among the localised enlargements (§ 189). 5. In *rachitic children* the liver and spleen may be pushed down by the deformity of the costal arches and so produce the appearance of an enlarged abdomen.

PART C. ABDOMINAL DISORDERS, THEIR DIAGNOSIS, PROGNOSIS  
AND TREATMENT.

§ 168. **Routine procedure and classification.** Having *first* ascertained that the patient's leading symptom is one of those above referred to, we *secondly* inquire into the history, and especially whether the condition came on acutely and suddenly, or is chronic and long-standing. The procedure to be adopted in acute cases, and that suitable in chronic cases, will be given under these respective headings. *Thirdly*, proceed to the physical examination of the abdomen, the routine method in ordinary cases consisting of (1) Inspection; (2) Palpation; (3) Percussion, to map out the boundaries of the liver, spleen, and other organs; and (4) Mensuration. In any doubtful case the rectum, vagina, urine, and fæces must certainly be examined.

If **severe abdominal pain**, which came on **suddenly** and acutely, be the leading symptom, first turn to § 169.

If **abdominal pain** of some duration and running a **chronic** course be the leading symptom, turn to § 175.

If there be a **generalised abdominal enlargement**, turn to § 183.

If there be a **localised tumour**, turn to § 188.



§ 169. **Acute Abdominal Pain**, coming on **suddenly**, includes amongst its causes some of the most serious conditions with which the physician or surgeon can have to deal; and on account of the large number of organs contained in the abdominal cavity, these causes include many pathological processes situated in various and often unsuspected positions.

The *causes* of abdominal pain may be conveniently classified for clinical purposes into nine groups:—

A. ABDOMINAL PAIN coming on **suddenly, with collapse.**

- |   |       |
|---|-------|
| I. Perforation of some organ or cyst (perforative peritonitis) . . . . .  | § 170 |
| II. Acute peritonitis due to causes other than the preceding  | § 171 |
| III. Acute Intestinal Obstruction (Hernia, Intussusception, Internal Strangulation and Appendicitis) . . . . .      | § 230 |
| IV. Displaced enlarged (or gravid) uterus; V. Embolism of the mesenteric artery; VI. Pancreatic Hæmorrhage. . . . . | § 172 |

B. ABDOMINAL PAIN coming on **suddenly, without collapse.**

- |   |       |
|---|-------|
| VII. Colic (Intestinal, renal, hepatic) . . . . .   | § 173 |
| VIII. Appendicitis (some cases); Floating Kidney; Splenic embolism; and some other obscure organic affections . . . . . | § 174 |
| IX. Visceral Neuralgiæ . . . . .  | § 174 |

In the first six the acute abdominal pain is usually ATTENDED BY COLLAPSE, but not in the last three. This however is only relative, and in any doubtful case the whole should be passed in review.

In order to ascertain which of these causes is in operation, and in view of the gravity of some of these cases, it will be desirable to consider the METHOD OF PROCEDURE in some detail.

(1) Regarding the *cardinal* or *leading* symptom, inquire carefully, as in all cases of "pain," concerning its position, character, degree, and intensity. The position of the pain is not always a guide to the organ affected, for it rapidly tends to become generalised; but the direction in which it is radiated is of great help in the diagnosis of the three kinds of colic. Whenever the three symptoms, ABDOMINAL PAIN, VOMITING, and CONSTIPATION, come on together suddenly, with COLLAPSE, the condition is very probably

due to either PERITONITIS (which may be due to PERFORATION), or INTESTINAL OBSTRUCTION.

(2) As to the *history of the illness*, it is useful to note if there had been any illness previous to the onset of the pain pointing to ulceration, dyspepsia, or other derangement of the abdominal organs.

(3) In the *examination of the patient*—(i.) his *age* is an important aid in the diagnosis of the cause of the pain. In childhood it is very probably some intestinal affection, such as colic, or intussusception; in adolescents and young adults, appendicitis, while cancer and tabetic crises may probably be excluded. In adults we think of hernia and ulcer of the stomach; in old age or after middle life we think of cancer, or if the patient is a female, biliary colic. (ii.) The *sex* may aid us, for in young females we may suspect an ulcer of the stomach even without previous symptoms; and in older women the rupture of an ectopic (extra-uterine) pregnancy, a condition which is frequently overlooked, or gall-stones. (iii.) The presence or absence of *tenderness* is of considerable aid. If it is quite absent peritonitis may be excluded, and its position will aid us in localising the lesion more than does the seat of the pain, as, for instance, MacBurney's point in appendicitis. (iv.) *All the organs* of the abdomen must be as carefully and as thoroughly examined as circumstances will permit. Never forget to examine per rectum and vagina, because stricture of the former or a pelvic tumour may throw considerable light upon the case. (v.) The patient's *general symptoms* must also be carefully investigated. If the temperature and the pulse be normal, we may exclude inflammatory conditions. The temperature alone is not a sufficient guide in this respect (see § 166), but in general terms no serious acute abdominal condition exists without the *pulse rate* exceeding 90 or 100. If the patient is much emaciated, in adults we must bear in mind obscure malignant disease, and in children the presence of tubercle.

If the pain, which is severe and has come on suddenly, is **attended by marked collapse**, first turn to § 170. If it is **unattended by collapse**, turn first to § 173. It must be remembered however that any severe pain will cause a certain amount of prostration.

I. *The patient complains of acute abdominal pain, which has come on suddenly, with symptoms of severe collapse, attended by vomiting and constipation; the pulse is rapid (over 100)—the case is probably one of three conditions, PERFORATION into the peritoneum, ACUTE PERITONITIS, or ACUTE INTESTINAL OBSTRUCTION.*

§ 170. **Rupture of a cyst or organ, or perforation of the alimentary canal** (which shortly develops into Perforative Peritonitis). The *cysts which may rupture* are hydatid or simple cysts of the liver, kidney, pancreas or other organs, ovarian and parovarian cysts, abscess of the liver, gall-bladder, kidney or other organs, and perityphlitic abscess (§ 176). *Rupture of an organ*, with consequent extravasation of blood, causes similar symptoms; and of such may be mentioned—ruptured fallopian tube (in cases of extra-uterine pregnancy), ruptured abdominal aneurysm, rupture of the liver or kidney (following injury). *Perforation of the alimentary canal* may at any time occur when an ulcer is present. These ulcers are, in order from above downward, simple ulcer of the stomach (which is usually met with in young æmemic women), simple ulcer of the duodenum (which occurs in males, or may follow burns), ulcer of the lower part of the ileum (due to tuberculosis or enteric fever), ulcer of the cæcum or appendix, ulcer of the large intestine, especially the sigmoid flexure (usually cancerous, dysenteric or syphilitic).

*Symptoms.* Pyrexia at first is absent, and the temperature may be subnormal because of the collapse. The pulse is thready, feeble, and rapid. The pain is probably very severe, and the ashen pallid face with its cold clammy sweat and sunken eyes are very characteristic. Vomiting is rarely absent; it may be incessant, distressing, and even stercoraceous. A certain amount of constipation is generally present on account of the paralysis of the bowel consequent on the pain, and it is only by this fact that these cases are distinguished from corrosive poisoning. Perforated *gastric ulcer* is perhaps the commonest condition of those above mentioned, and may be taken as a type. We should inquire for a history of dyspepsia and other symptoms (§ 208), but in not a few cases rupture has occurred without previous symptoms of any kind whatever. On examination there is tenderness, most marked in the epigastrium, rigidity of the muscles, and a tympanitic note

over the whole abdomen.<sup>1</sup> After a few hours there is a deceptive period of repose, during which all symptoms of discomfort are diminished. On examination several hours later, however, symptoms of collapse with acute peritonitis (§ 171) are found, *generalised*, in a case where there were no adhesions at the site of rupture, or *localised*, where adhesions were present. The symptoms of perforated *duodenal ulcer* may be the same as those of gastric ulcer, but the condition occurs usually in men. The symptoms of perforation of another part of the intestine, or rupture of a cyst, are much the same, and one can only hazard a diagnosis as to its situation by the site of the pain and tenderness, and the previous history. There are 3 degrees of severity met with when perforation of the intestine occurs—(a) When there are adhesions the peritonitis may be localised or partial; (b) when there are no adhesions, but a small leakage, it may be only moderately sudden in its onset; (c) when the leakage is large it will be extremely sudden and severe in its onset.

The *latent period* which ensues shortly after an acute onset deceives many clinical observers. The pain may subside, all symptoms decrease, and the temperature become normal or sub-normal. But (1) the *pulse remains persistently high*, and (2) in blood films the polynuclear cells, and perhaps the total leucocytes, will be markedly increased; and these are sufficient to indicate immediate exploratory abdominal section. Perforative peritonitis may have to be diagnosed from diaphragmatic pleurisy and pneumonia of the base, where the pulse-respiration ratio is disturbed, but not the pulse-temperature ratio.<sup>2</sup>

*Treatment and Prognosis.* Laparotomy should be performed at once, this being now advised by surgeons in nearly all cases of supposed rupture of an organ, if only as an exploratory procedure. If deceived by the period of repose into thinking the patient is recovering, in a few hours general peritonitis will have set in and operative interference will be too late. In cases where the patient has been operated upon within the first 12 hours 79 per cent. recover; if after 24 hours, only 29·4 per cent. recover (Goffe). The after

<sup>1</sup> The disappearance of the liver dulness in a case presenting these symptoms has been considered pathognomonic of ruptured gastric ulcer.

<sup>2</sup> See Report of Clin. Soc. Lond., *The Lancet*, April 19, 1902.



treatment depends on the cause. In the case of rupture consequent on injury, internal hæmorrhage takes place with a fatal result, but even in such cases laparotomy has been performed with satisfactory results.<sup>1</sup>

II. *The patient complains of SEVERE ABDOMINAL PAIN, EXTREME PROSTRATION, and VOMITING; there is THORACIC RESPIRATION, and the TEMPERATURE IS ELEVATED—the disease is ACUTE PERITONITIS.*

§ 171. **Acute Peritonitis** (General Peritonitis) is an acute inflammation of the peritoneum. It is rarely, perhaps never, a primary disease, but its onset is usually sudden.

*Symptoms.* (1) The aspect is very characteristic; the countenance has an anxious pinched look, the cheeks flushed, and the skin cold and clammy. (2) The pain is severe and constant, but liable to exacerbations on account of the intestinal peristalsis and the passage of wind along the bowel.<sup>2</sup> It is also increased by any kind of movement, even by the respiratory movements. Consequently (3) the respiration is thoracic, and careful inspection will show that (4) the abdominal walls are immobile and rigid. There is acute tenderness on pressure, so much so that the weight of the bed-clothes can hardly be borne. (5) The posture of the patient is very characteristic, as he lies on his back with legs drawn up to relax the abdominal muscles. (6) Pyrexia, ushered in suddenly with rigors, and attended by a small, wiry, rapid pulse of 100 to 140 per minute. The temperature is elevated only two or three degrees above normal, and maintained there continuously, unless a pyæmic process be present, in which case there are rapid variations of wide range. In some cases, *e.g.*, perforation, it may be subnormal at first (*vide supra*). There is marked prostration, as in all abdominal inflammations, and a great tendency to collapse, even from the beginning. (7) The bowels are constipated, and there is persistent vomiting. Hiccough is often present, and if persistent is a very bad sign, as in all abdominal disorders. There is diminution of urine, and this may amount to suppression. The urine is abundantly charged with indican. Death occurs from collapse or asthenia, and the mind remains quite clear

<sup>1</sup> *E.g.*, in cases of ruptured kidney—Bland Sutton, Clin. Soc. Trans. 1899–1900.

<sup>2</sup> The acute peritonitis which complicates enteric fever is of a latent character and unaccompanied by pain. This and puerperal peritonitis are the only exceptions.

until the end in uncomplicated cases. Peritonitis is practically never a primary affection, and careful inquiry should reveal the cause.

The *causes* of acute peritonitis may be grouped under 6 heads : (i.) *Injury or operation*. In cases occurring in women without obvious cause, the possibility of criminal procedure for abortion should always be remembered. As regards surgical operations on the belly, modern experience has shown that it is not so much the actual injury as the introduction of septic organisms, which produce the peritonitis ; and that if these be excluded, mere damage to the peritoneum will not cause a generalised peritonitis. (ii.) *Extension* of inflammation from the thorax, or from various organs of the abdomen, *e.g.*, appendicitis, gonorrhœal salpingitis, inflammatory conditions of the intestines (typhoid, dysenteric and other). (iii.) *Poisons in the blood* of various kinds, *e.g.* streptococcal, staphylococcal, and gonorrhœal infection. Peritonitis is apt also to complicate scarlatina, pyæmia, and the other acute specific fevers. *Puerperal* peritonitis is due to the introduction of a pyogenic infection through the raw uterine surface. A chronic form of the disease arises in *uræmia*. (iv.) *Idiopathic peritonitis* was the name formerly given to the disease when no cause could be discovered. But there are good grounds for believing that such cases are due to the entrance of a specific poison of microbic origin into the blood, probably through the intestinal wall previously rendered pervious by damage or disease. (v.) *Chill*, under certain conditions, such as bathing during the menstrual period, is sometimes included as a cause, though this usually leads to a chronic localised peritonitis (perimetritis). Apart from this it seems probable that the true explanation of peritonitis after a "chill" is to be found under heading (iv.), *viz.*, microbic infection. (vi.) *Rupture* of some abdominal cyst, such as ovarian cyst, an abscess of the liver, or rupture of the gall-bladder, &c. (see § 170). Rupture of a Graafian vesicle may give rise to a monthly peritonitis, but this is usually localised and less serious. (vii.) *Perforation of some part of the alimentary canal*, which had previously become thin by ulceration—ulceration of the appendix vermiformis, simple ulcer of the stomach (malignant ulcer rarely or never perforates because of the infiltration around), typhoid ulcer of the ileum, &c. (see PERFORATIVE PERITONITIS).

Acute peritonitis has to be *diagnosed* from four diseases:— (1) Acute intestinal obstruction, in which the constipation is absolute and no flatus is passed; there is usually no pyrexia, and the constitutional disturbance is usually less. (2) In *colic*, although the pain is also very severe, there is an absence of tenderness, and pressure may give relief. Pyrexia and collapse are absent, and the pulse is normal. (3) In *catarrhal enteritis* there is pain, and there may be vomiting and tenderness on pressure, but in this disease there is profuse diarrhœa. (4) In certain cases of *hysteria*, acute peritonitis may be very accurately simulated, though the temperature is normal, there is very little collapse, and there are evidences of the hysterical stigmata.

*The prognosis* is always very serious. As regards etiology, perforative peritonitis, formerly considered the gravest, is probably now the most hopeful if promptly dealt with. Modern surgery has done much for the rescue of such cases, and undoubtedly the most favourable of them is that due to appendicitis. Cases of this disease, if properly managed, should hardly ever be lost. The prognosis in any particular case depends, in general terms, on the cause, and the severity of the collapse, the dyspnoea, and the hiccough.

*Treatment.* The indications are (1) to allay the inflammation, and (2) to remove the cause.<sup>1</sup>

(1) Various antiphlogistic measures are called for, but purgatives should be avoided. Blood-letting used to be recommended, though some disapprove on account of the collapse. Great relief is sometimes obtained by 10 or 12 leeches on the abdomen. Absolute rest, the patient not being allowed even to turn in bed, is essential, especially in perforative peritonitis. Local applications may allay the pain and the inflammation. Cold, in the form of an ice bag, is more useful in arresting the disease, though heat is efficacious in relieving pain. Opium, from a medical point of view, is the remedy which does most good, as it not only relieves the pain, but it reduces the peristalsis of the bowel, and so brings about that

<sup>1</sup> Much valuable work has been done of late years in the life history of the microbes referred to above under causation. When we possess the means of identifying the peccant microbe in any given case of peritonitis, a corresponding antitoxin will probably also be available. An antistreptococcal serum is already at command.

perfect rest which is essential to recovery. It is surprising what large doses will be tolerated; gr. i. or ii. of the extract every two or three hours, if the patient be awake, may be taken. It should be given always in a fluid form.<sup>1</sup> It generally allays the vomiting; if it fail, give it subcutaneously. Surgeons, however, avoid opium, because it masks the symptoms. The diet should consist of easily assimilated fluid nourishment, cold, with moderate quantities of stimulant, according to the state of the pulse; spoonfuls of beef-tea and milk alternately, with brandy. The hiccough may be relieved by sucking ice, spt. æth., opium, or chloral. Prompt surgical measures are called for in many cases.

III. *The patient complains of acute abdominal pain, which is attended by collapse, and the PULSE IS RAPID; there is ABSOLUTE CONSTIPATION with inability to pass even flatus, and VOMITING (at first of food, then of bile, and finally of stercoraceous matter)*—the condition is ACUTE INTESTINAL OBSTRUCTION.

**Acute Intestinal Obstruction**, i.e., obstruction coming on suddenly, is always a matter of serious importance, and every practitioner should be thoroughly acquainted with its several causes. In actual practice, whenever the three symptoms, **constipation, vomiting and abdominal pain**, occur together, one of three conditions should be suspected—acute peritonitis, intestinal obstruction, or colic.

The various causes of acute intestinal obstruction—the chief of which are External Hernia, Internal Strangulation, Intussusception, and Appendicitis—are fully dealt with under intestinal disorders (Chapter XI.), Appendicitis being described below (§ 176).

§ 172. *The patient complains of acute abdominal pain with more or less collapse, the temperature is probably normal or subnormal, but the symptoms do not quite conform to any of the preceding—some of the rarer causes are probably in operation, such as the following:—*

VI. **Displacement of a Gravid Uterus** is known by the pain being referred to pelvis, and examination revealing the local mischief. It may occur when jumping from a height, and performing active exercise, especially in early pregnancy (Chapter XIV.).

V. In **Embolism of the mesenteric artery**, a cause of embolism, such as endocarditis, is present. It is rarely diagnosed during life. The absence of symptoms pointing to the other causes may lead one to suspect embolism.

<sup>1</sup> I have several times found a large number of opium pills in the stomach and intestines after death in such cases.



**VI. Acute pancreatitis.** (1) The pain here is very sudden and severe, usually in the upper part of the left side of the abdomen; (2) vomiting and constipation are usually present; and (3) there is usually tympanitic abdominal distension (see also § 182).

**Vla. Hæmorrhage into the pancreas** is attended by (1) severe and sudden pain in the upper part of the abdomen; and that part soon becomes tender; (2) vomiting of increasing severity; (3) symptoms of collapse, with the restlessness and subnormal temperature which accompany collapse when it is due to hæmorrhage (§ 182).

Very few cases of VI. and Vla. have been recorded<sup>1</sup>; and the *diagnosis* of both of these conditions from intestinal obstruction or perforation into the peritoneum is usually impossible before laparotomy is performed. In both diseases death from collapse is the usual result. In acute pancreatitis death occurs usually in 4 days.

**VII.** *The patient complains of acute abdominal pain, which has come on suddenly while apparently in good health without definite collapse; the pulse does NOT EXCEED 100; there may be VOMITING and constipation*—the case is probably one of the 3 kinds of COLIC, though APPENDICITIS, VISCERAL NEURALGIE, and some OTHER AFFECTIONS may come on in this way.

§ 173. Colic is a somewhat vague term applied to spasmodic paroxysmal pain situated in the abdomen. There are three kinds—**intestinal, hepatic, and renal colic**—and they have the following features in common:—(1) the pain is extremely severe, and sudden in its onset; (2) not infrequently there is vomiting, from the severity of the pain; (3) the face is pale and “anxious,” and in severe cases the pulse is rapid and feeble, though it practically never exceeds 100; (4) *the temperature is neither above nor below normal*; (5) the physical signs in the abdomen are negative, and the pain may even be relieved by pressure.

(a) **Intestinal colic** is due to spasm of the bowel caused by the irritation of masses of hard pieces of undigested food, such as raw apples, etc. The pain of intestinal colic is characteristically twisting, paroxysmal, and limited to the abdomen, principally around the umbilicus, and is relieved by pressure, which distinguishes it from peritonitis. The abdomen may be distended with flatus. Sometimes it is followed or accompanied by diarrhœa, or, as in lead colic, by constipation. The pain of colic due to lead-poisoning is accompanied by a slow and hard pulse, with contraction of the abdominal muscles and other symptoms, such as a blue line on the gums; and a history of working amongst lead is obtainable.

(b) **In hepatic colic**, which is due to the passage of gall-stone into the duodenum (*q.v.*), the pain shoots *upwards* to the right shoulder and backwards, never downwards; a dull pain continues

<sup>1</sup> See Middleton Goldsmith Lecture, *New York Medical Record*, Vol. I., 1889.

during the intervals between the spasms. After lasting a few hours or a day or two, it is followed by jaundice.

(c) **Renal colic** is due to the passage of a calculus along the ureter. The pain radiates *downwards* from the loin to the thigh and the testicle of the same side, which is often retracted. It may last for a day or two. During the attack micturition is frequent, sometimes there is hæmaturia or strangury. There will probably be a history of gravel in the urine, or attacks of a similar nature.

The *diagnosis* of intestinal from other forms of colic is given in Table XV. below.

TABLE XV.—DIAGNOSIS OF COLIC.

	Character and distribution of pain.	Associated symptoms.	Age and sex of patient.
<b>Intestinal.</b>	Twisting, around umbilicus, paroxysmal; relieved by pressure.	Constipation (or diarrhœa). No jaundice.	Any age or sex. Sometimes evidence or history of plumbism.
<b>Biliary.</b>	In right hypochondrium shooting upwards to right shoulder, constant, but also in paroxysms.	Jaundice soon supervenes. Other hepatic symptoms may be present.	Female sex. At or after middle life.
<b>Renal.</b>	In loin, shooting down to thigh and testicle or ovary of same side.	Crystals or other urinary change, hæmaturia. No jaundice. Sometimes frequent micturition or strangury.	Usually male. Children and adults.

*Prognosis.* The course of an attack of colic is short and severe. If the patient does not recover in the course of one or two days, we must suspect one of the more serious conditions mentioned on p. 301. The pulse must be carefully watched.

*Treatment.* For all forms of colic some of the following measures—hot fomentations, a warm bath, belladonna, opium, or chloroform, as local applications, and hypodermics of morphia (gr.  $\frac{1}{6}$ , with atropin gr.  $\frac{1}{60}$ )—may be necessary to alleviate the extreme pain. Large draughts of warm water should be taken. For intestinal colic in particular, a full dose of castor oil with 20 minims of laudanum should be given, followed by saline purgatives. If it is due to lead poisoning, start a course of treatment with small doses of potassium iodide and mag. sulph; and for prevention, scrupulous cleanliness and the systematic drinking of lemonade containing sulphuric acid are advisable. Hepatic and renal colic are treated under gall-stones (§ 243) and renal colic (§ 302).

§ 174. VIII. Among the **rarer causes of abdominal pain** without collapse are various **OBSCURE ORGANIC AFFECTIONS** of the abdomen, evidenced at first only by pain. Two may be mentioned which came under my notice, **PANCREATIC CALCULUS** and **OBTURATOR HERNIA**, in both of which the only symptom for some time was pain coming on **SUDDENLY** without collapse. In the former the pain was extremely severe, and of a paroxysmal character, situated just below the umbilicus; later on it was associated with fat in the fæces, emaciation, and glycosuria.

**DISLOCATED** or **FLOATING KIDNEY** (§ 178), which is a more frequent condition than is usually supposed, may be attended by a constant (chronic) pain: or give rise to severe attacks hardly distinguishable from intestinal colic.

**APPENDICITIS** is also a cause of abdominal pain which may be of sudden onset. Sir William Macewen told me of the case of a young man who was *suddenly* seized with severe abdominal pain in jumping out of a hansom cab (see also footnote on p. 314). But appendicitis is more apt to be a chronic disease, and is therefore treated of more fully in § 176.

In **SPLenic EMBOLISM** the pain is generally sudden in onset, but is not usually very severe or lasting, and is referred to the splenic region. Its most common cause is acute or chronic endocarditis, evidences of which are present (§ 42).

In most of the obscure organic affections the pain comes on gradually, and is of a chronic character.

IX. In **Visceral Neuralgiæ** abdominal pain may come on suddenly and acutely, and it may be for a long time the only symptom.

(1) *Gastralgia* or gastric neuralgia is rare, but it is the most typical and best known visceral neuralgia. The pain is severe, periodic, but usually relieved rather than aggravated by food or by pressure. The skin may, however, be very sensitive to the flick of a handkerchief (§ 207).

(2) The gastric crises and neuralgia of the bladder or other viscera in association with *tabes dorsalis*.

(3) *Neuralgia* or "colic" of other viscera (*i.e.*, pain in the viscus without functional or organic derangement) have been described by various authors—*e.g.*, ovarian colic, vesical or splenic neuralgia, etc.; though these cases sometimes turn out to be connected with an undiscoverable organic disease or with *tabes dorsalis*.

(4) The neuralgia which accompanies or follows *herpes*.

(5) *Angina pectoris* is in some cases referred more to the abdomen than it is to the chest, but it is recognised by the circulatory disturbances, etc. (§ 44).

(6) *Migraine* is certainly met with sometimes alternating with abdominal pain.

§ 175. **Chronic abdominal pain** implies that kind of abdominal pain which has come on somewhat gradually and is running a chronic course. Chronic abdominal pain may be produced by a large number of causes which it would be impossible even to enumerate. It is only possible here to refer to those conditions which are likely to be obscure by reason of their **NOT PRESENTING** signs or symptoms distinctly pointing to some affection of

the STOMACH, LIVER, SPLEEN, or other ABDOMINAL VISCUS; these latter conditions being dealt with in the following chapters.

I. Appendicitis	§ 176
II. Chronic Intestinal Obstruction (Malignant Stricture, Simple Stricture, Pressure by a tumour, Paralysis of the bowel, etc.)	§ 231
III. Chronic Peritonitis	§ 177
IV. Movable Kidney	§ 178
V. Intestinal Dyspepsia; VI. Enteroptosis; VII. Obscure visceral and spinal Disease; VIII. Pancreatic disease §§ 179—182	

The history must be thoroughly investigated, and every organ thoroughly examined. Three things may afford us important clues—

1. The POSITION, character, degree, and constancy of the *pain*, and the presence of *tenderness* must be observed. (i.) If the pain and tenderness be *generalised*, one might suspect Tubercle or Cancer of the Peritoneum. (ii.) If they be situated chiefly in the *lower abdomen*, one might suspect Appendicitis or incipient disease of the Bladder or Uterus. (iii.) If the pain be chiefly in the *upper abdomen*, incipient Gastric or Liver disease. Thorough and REPEATED EXAMINATIONS of the *abdomen*, *rectum* and *vagina* are nearly always necessary. The *urine* also should be repeatedly examined for gravel, etc., and the *feces* (§ 217) for gall-stones. If there be general abdominal enlargement, turn to § 183; if a localised tumour, turn to § 188.

2. The AGE of the patient, and the history and duration of the illness, should be inquired into. In *children* perhaps the commonest of the obscure causes of chronic abdominal pain are intestinal worms and tuberculosis of the peritoneum; in the *aged* incipient cancer of some organ.

3. The STATE OF THE BOWELS both previously and at the time of examination. In I. and II. above there is constipation; while in most of the other causes there is diarrhoea or irregularity of the bowels.

*The abdominal pain is constant but liable to EXACERBATIONS, especially after exercise; there is TENDERNES in the left inguinal region; the PULSE is RAPID, and the TEMPERATURE elevated from time to time; the patient is young—the disease is probably APPENDICITIS.*

§ 176. **Appendicitis** (typhlitis, perityphlitis) is a disease now known to be much more common than used to be suspected, yet it is still frequently overlooked. Appendicitis may consist simply of a *catarrhal* inflammation of the vermiform appendix, which may go on to *ulceration*, *peritonitis* (usually localised), or *perforation*. It is originated by an impaction in the appendix of intestinal concretions, undigested food, or some other foreign substance such as a fruit stone or tooth-brush bristle; any of which can, as



Mr. Lockwood has shown, start a highly septic inflammation.<sup>1</sup> Inflammation may extend to the cæcum (typhlitis) or the surrounding tissues (perityphlitis). Adhesions frequently form round the appendix, due to a localised peritonitis. If the appendicitis in such a case goes on to perforation, a *local peritonitis* or *local abscess* results. This abscess may burst into the general peritoneal cavity, but more often burrows extra-peritoneally in various directions; thus it may form a perinephric abscess, or may open externally over Poupart's ligament. In other cases septicæmia or pyæmia may occur. Both intra- and extra-peritoneal abscesses may open into the bowel, or bladder. Sudden perforation of the appendix, without previous formation of adhesions, or after the breaking down of adhesions, results in *generalised peritonitis*.

There are two clinical forms of appendicitis: Acute or recurrent, and chronic appendicitis. (a) In CHRONIC APPENDICITIS there may be no symptoms other than pain in the right iliac region increased after any over-exertion. Sometimes there is also alternating diarrhœa and constipation: there may or may not be local signs of swelling or tenderness.

(b) RECURRENT APPENDICITIS consists of recurring acute attacks. Here again the course of the disease is essentially a chronic one, with a constant liability to a recurrence of the inflammation. After this has subsided the patient may go on for many months in apparent health. Possibly, he may never be troubled again, but in the vast majority of cases a fresh attack of inflammation occurs sooner or later, with localised, and sometimes generalised peritonitis.

*Symptoms.* In a typical *attack of appendicitis* there are 4 symptoms, which, occurring in a young person, point to appendicitis—pain with tenderness, constipation, gastric symptoms, and slight pyrexia. (1) The chief symptom, as above mentioned, and sometimes the only one, is pain with tenderness, usually situated in the right iliac region. The *tenderness is generally fixed*, and is nearly always in this situation; but the *pain has a tendency to radiate*, and it may be referred to the umbilical, or even to the left inguinal region. It is a constant, dull, aching pain, liable to be brought on or *increased by exertion*. The pain generally comes on quite

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<sup>1</sup> "The Pathology and Treatment of Appendicitis," by C. B. Lockwood; Macmillan, London, 1901.

gradually, but sometimes suddenly.<sup>1</sup> Special tenderness is present at "MacBurney's point"—*i.e.*, midway between the umbilicus and the right iliac spine. (2) Deep-seated local swelling is sometimes absent; but there is usually a feeling of resistance, with dulness to percussion, or an indefinite tumour, in the r. iliac fossa. (3) Vomiting may be urgent at the onset of an attack; when it continues for many days the prognosis is unfavourable. Constipation is usually present, so that the case is apt to be mistaken for intestinal obstruction. In other cases the attack is ushered in with diarrhoea. The urine is scanty, and the bladder irritable. (4) The pulse is quickened, and the temperature rises soon after the onset of pain, and remains about 100° to 102° for a few days (see Fig. 106, § 385). The pulse forms the best single indication as to the acuteness of the progress.

*Course and Prognosis.* When an acute attack, as above described, sets in, there are three possible events—recovery, local abscess formation, or general peritonitis. (1) In a favourable case the temperature falls about the third day, the swelling disappears, pain and other symptoms subside, and the patient may be well in ten days. In other cases slight fever persists for a few weeks, and there is an indurated swelling left, due to adhesions. The patient may go about for months or years with chronic appendicitis, and apart from vague pains, general malaise and dyspeptic symptoms suffer no inconvenience. At any time, however, he is liable to have a recurrence of the acute symptoms. (2) When the general symptoms show no improvement by the third day, and the local swelling progressively increases, it is probable that an abscess is forming. (3) Perforation, with generalised peritonitis, may occur at any time. The general symptoms in such cases are much more severe, vomiting persists, and the abdomen is distended and motionless by the second or third day.

*Treatment.* Rest in bed and light diet are essential. Hot fomentations locally are useful for the pain. Opium in small doses (short of causing drowsiness) is also admissible for the relief of pain, after the diagnosis is established. It should not be given for

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<sup>1</sup> I have recently seen a case where the advent was so sudden that the pain could only be diagnosed from intestinal colic by the rapidity of the pulse, slight elevation of temperature (99.6° at night), and by the duration of the pain for several days, after the bowels had been thoroughly relieved.

long, as it not only confines the bowels, which is injurious, but masks the symptoms on which surgeons rely as indications for operation. Other hypnotics may be employed. An enema may be given on the third day if the condition be improving.

The *question of operation* requires careful consideration, and a surgeon should be early in touch with the case. Mr. Lockwood's rules (*loc. cit.*) are as follows. During the first few days of an acute appendicitis the patient must be carefully watched, and if symptoms of perforation with collapse or general peritonitis set in, surgical aid must be promptly sought. If there are signs of intestinal obstruction, but the patient can pass flatus, and the pulse remain under 100, watch the case, but be ready to operate at any moment. When flatus cannot be passed, and the pulse is over 100, or when there are signs of abscess formation, operation is called for at once. In chronic appendicitis the bowels must be opened regularly; if care be taken the disease may remain quiescent. Yet the question of operation must be entertained even in these cases, as there is a constant danger of relapse and perforative peritonitis.

*In addition to chronic abdominal pain, there is a history of CONSTIPATION, gradually increasing to COMPLETE STOPPAGE of the bowels, with the gradual supervention of VOMITING*—the case is probably one of CHRONIC INTESTINAL OBSTRUCTION.

In **chronic intestinal obstruction** (§ 231) the abdominal pain is more or less generalised and intermittent. The constipation may at first have alternated with diarrhœa, but after a time it is so complete that not even flatus can be passed. Vomiting, at first of food, and later fæculent matter, a rapid pulse and other constitutional symptoms ensue if the condition is not relieved. The four commonest causes are Malignant Stricture, Simple Stricture, Pressure of a Tumour, and Paralysis of the lower bowel.

*The abdominal pain is chronic and GENERALISED; it is attended by CONSTITUTIONAL SYMPTOMS, and some ABDOMINAL ENLARGEMENT or other local signs; the patient is under or over middle age*—the disease is probably CHRONIC PERITONITIS.

§ 177. **Chronic peritonitis** runs a slow and chronic course, and it is mostly attended by a certain amount of generalised pain. Some authorities describe a simple or idiopathic chronic peritonitis, but practically the disease is only met with in two forms: (a) that due to the deposit of **tubercle**, and (b) that which attends the deposit of **cancer**—two conditions which, by the way, are met with at the opposite extremes of life, and which present a very marked contrast both in their clinical and anatomical features.

In **chronic tubercular peritonitis** (tuberculosis of the peritoneum, abdominal tuberculosis) the patient is nearly always a child. There is a deposit of tubercle in the subperitoneal tissues, and the intestines become *matted together by adhesions*. Sometimes fistulous openings form between different portions of the bowel. (1) Pain and tenderness are present, but are not very marked features excepting during one of the subacute or acute exacerbations which arise. (2) Tubercular peritonitis is contrasted with cancerous p. by the marked tendency to the formation of adhesions without fluid, while in cancerous p. there is effusion of fluid without adhesions. As a consequence of the adhesions and the deposit of tubercle, knots or thickenings can be felt through the abdominal walls, which have a very characteristic doughy or "boggy" feeling. (3) Fluid may be present when the disease occurs in young adults; and in acute cases. As the disease advances there is considerable tympanitic distension. (4) There is emaciation and hectic fever—*i.e.*, morning temperature normal and an evening rise of two or three degrees, as in all active tubercular processes. (5) Tubercle is generally found in other parts also, especially in the lungs. (6) In one form of this disease in children a tubercular abscess forms, which points at the umbilicus, and gives rise to a *persistent discharge from the navel*.

*Prognosis and Treatment.* The prognosis is very serious, though not so bad as formerly. Much may be done by treatment on the same lines as for pulmonary tuberculosis, with the administration of sedatives if much pain be present. Operative measures have been successfully carried out in an early stage of the affection, by simply opening the abdomen. The practice of washing out the peritoneum with weak perchloride (say, 1 in 2,000) is now almost abandoned. Plain water seems equally successful; and it is more successful if done early; indeed it is only justifiable before adhesions have become firmly established. Much may be done by the diligent emunction with blue ointment (ung. hyd. B. P.).

**CHRONIC CANCEROUS PERITONITIS** (Cancer of the peritoneum) is always attended by a great deal of pain, constant but also in paroxysms. There is a great tendency to the rapid formation of



fluid in the abdominal cavity, which is nearly always tinged with blood. It arises only in late middle or advanced life. Its recognition is easy in typical cases, on account of the age, acute pain, and ascites (under which heading it is described, § 186).

*The pain is of a "dragging" character, INCREASED BY EXERTION, accompanied by DYSPEPTIC and other VAGUE SYMPTOMS*—the disease is possibly **DISLOCATION OF THE KIDNEY**.

§ 178. **Movable Kidney** (also called Dropped, Dislocated, or Floating Kidney, according to the degree of mobility). Suckling, of Birmingham,<sup>1</sup> states that of 100 women and 100 men whom he examined consecutively in the course of a few weeks, in 42 of the former, 26 of the latter, the kidney was unduly movable. Other authors have not found so large a prevalence, but a slight degree of dislocation is more common than is usually believed. In women the r. is more frequently movable than the l. kidney, because of the anatomical relationship of the former with the liver; whereas in men both kidneys are equally affected.

The *physical signs* of this condition can only be discovered by palpation of the abdomen, with the patient lying down. The method of palpating the kidneys is given in § 294. With the patient in the erect or sitting posture the kidney comes down more during inspiration than when lying down. After a little practice the patient will be able to lean forward and relax her muscles, which is an important aid to the observer. The left kidney rarely falls below the umbilicus, but the right one may be displaced into the iliac fossa, and even into the pelvis.

*Symptoms.* One or both kidneys may be movable without giving rise to any symptoms, but in the majority of cases there is pain of a very characteristic "dragging" kind and marked ill-health. When the right kidney is movable there is not so much pain as an uneasy feeling on the right side, back and front. In the case of the left kidney, pain is very severe, but disappears when the patient lies down. It is renewed, however, on sitting, standing, or walking. Sometimes severe attacks of "colicky pain" occur. Dropped kidney may be accompanied by a great number of other symptoms, such as mental depression and hypochondriasis,<sup>2</sup> a constant feeling of exhaustion, difficulty in walking, standing, or even sitting upright, disturbance of the liver, palpitation, vertigo, dyspepsia, albuminuria, constipation, or diarrhœa.

*Etiology.* The fact that a very much larger percentage of women than of men have movable kidney is attributed by Suckling to the wearing of corsets. A fall or strain will also displace the organ, and that is why it

<sup>1</sup> On "Movable Kidney," 1898.

<sup>2</sup> Many and various are the nervous symptoms referable to a dislocated or movable kidney. A lady æt. 56 recently consulted me for neurasthenia, from which she had suffered for many years. She was extremely depressed and melancholic, and had many nervous symptoms. On inquiry I found she had for 10 or 12 years suffered from severe "colicky pains" in the abdomen, and from time to time very severe attacks of pain which had been variously diagnosed by different doctors, as biliary, renal, and intestinal colic. She had a constant dragging pain in the back and abdomen, and was only easy when lying down with the legs bent or sitting up in a forward position supporting the lower abdomen on the palms of her hands. I found her left kidney dropped nearly to the brim of the pelvis; on wearing a properly adjusted abdominal belt nearly all her symptoms disappeared.

is advisable for those with spare abdominal muscles to wear a belt when at work in the gymnasium. Attendants at refreshment bars, who have to draw beer or draw corks, are often found to suffer from movable kidney. It is said to be extremely common among those who suffer from migraine (owing possibly to the retching, which is a feature of that condition); and it occurs more often in tall than in short people.

*Treatment.* Bromides and rest will relieve the patient for a time, and any concurrent dyspepsia must be remedied; but the radical treatment consists either in the wearing of a proper form of belt, or an operation for stitching up and attaching the kidney in position. The abdominal belts usually supplied by instrument makers are not very successful; but Suckling has designed an apparatus<sup>1</sup> for applying additional pressure outside the belt, which he claims does away with the necessity of operation, and is permanently successful. Tonics and fattening of the patient may be tried, but in my experience they are not very successful.

*Among the rarer causes of chronic abdominal pain may be mentioned VISCERAL NEURALGIÆ (§ 174), INTESTINAL DYSPEPSIA, ENTEROPTOSIS, INCIPIENT SPINAL or VISCERAL DISEASE, and DISEASE OF THE PANCREAS.*

§ 179. **Intestinal Dyspepsia and Intestinal Catarrh** are conditions which it is sometimes a little difficult to separate from one another and from gastric dyspepsia, and some doubt whether they ought to be described as separate entities. The patient generally complains of obscure and erratic pains in different parts of the abdomen. He also complains of irregular attacks of diarrhoea and constipation, brought on by very slight dietetic errors or exercise. There is generally a good deal of flatus passed per rectum. The faeces are offensive, very often fermenting and decomposing, and *contain a good deal of undigested food*. This latter really constitutes the ground for regarding the disease as a separate one. When there is rectal or intestinal catarrh there is a certain amount of mucus (see § 217) and specks of blood (not streaks, such as come from piles) in the faeces. The patient also generally complains of a feeling of exhaustion, with little or no exertion, and there may be prostration, nervousness, and other nervous symptoms. In very prolonged cases there is loss of flesh, and the appetite is capricious. The disease in itself is not very serious, though it is inconvenient and intractable. In view of the large number of organisms normally found in the intestinal contents, any continual damage of the intestinal wall, such as intestinal catarrh, is to be deprecated, since the bacilli can then more readily make their way through a damaged wall, and in this way give rise to what used to be called idiopathic peritonitis and other troubles.

*Treatment.* The indications are:—(1) To prevent the decomposition which goes on in the intestines. (2) To allay the irritation of the bowels. Dietetic measures are generally called for, and the patient may derive benefit from diet consisting entirely of boiled milk, for a time; and the water which he takes should always be boiled. The bowels should never be allowed to be confined. If there is a tendency to constipation an occasional dose of hyd. cum cret. or castor oil (which can be given in capsules), or salines, is useful. Intestinal antiseptics are

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<sup>1</sup> Supplied by Messrs. Sall, of Birmingham.

very beneficial, such as salol, menthol, thymol, and eucalyptus, which are not absorbed by the stomach. Enemata containing these drugs may also be given.

§ 180. **Enteroptosis** (syn. visceroptosis, Glénard's disease, abdominal ptosis, dropping of the viscera) is a condition in which there is a general ptosis, or downward displacement or dropping of one or more of the movable abdominal viscera. Any of the viscera may, it is believed, be dropped in this way, owing to laxity of their ligaments or mesentery—liver, spleen, kidneys, and even intestines (Glénard). Ptosis of the liver may be mistaken for floating kidney. Another and rarer condition may be similarly mistaken, namely, a floating lobe of the liver. Dislocated kidney is the one most frequently recognised (§ 178); but any viscus may be "dropped." It is generally associated with certain nervous symptoms, resembling neurasthenia. The precise interpretation of these local and general symptoms and their association with one another is still uncertain.<sup>1</sup> The most constant *symptoms* are: (1) Pain or a sense of weight or dragging in the abdomen, and sometimes in the back, accompanied by a feeling of sinking, or of emptiness or hollowness, is constantly present; from time to time the pain may assume a colicky character. It has been noticed in many cases that there is tenderness at a localised spot a little to the l. of the middle line, just above the level of the umbilicus. (2) There is generally nausea, and from time to time vomiting. Symptoms of dyspepsia are usually present. (3) Sometimes there is diarrhoea, but more often constipation, and it is a special feature that aperients seem to cause considerable distress. (4) Great depression, nervousness, a general unfitness for all forms of exertion, and indeed all the symptoms of neurasthenia, may ensue; and the patients are apt to drift gradually into hypochondriasis. It will be remembered that nervous symptoms such as these were associated with movable kidney (§ 178). (5) The examination of the abdomen should be made while the patient is standing erect. The position of the viscera should then be marked; and afterwards, an examination should be made with the patient in a recumbent posture. In marked cases the symptoms are considerably aggravated by the erect position, and they may be relieved by lying down, by pressing on the lower abdomen, or by wearing a supporting belt. Undoubtedly in many cases a displacement of the viscera can thus be made out, and when the patient is upright a normal sized liver, or even a kidney, may be mistaken for a tumour. It is a curious fact in this connection that certain persons may have this displacement of organs, and yet do not suffer from any of the above symptoms.<sup>2</sup>

The *prognosis* as regards recovery is very uncertain. On the other hand, the condition is not fatal, and much can be done by judicious treatment if the physician secures his patient's confidence. The lives of these patients are often very miserable.

*Treatment.* The indications are: (1) To relieve the nervous symptoms; (2) to regulate the excretories; (3) to support the viscera. Much relief

<sup>1</sup> A large proportion of the nervous symptoms belong to the vaso-motor system, and all of them can be explained by a presumed derangement of this system (see "Clinical Lectures on Neurasthenia"). Bearing in mind the large part which the great reservoir of blood in the splanchnic vascular system must play in the vaso-motor mechanism; bearing in mind also that the great central sympathetic ganglia are situated within the abdominal cavity; one can readily understand that derangement of the sympathetic nervous system should be associated with dislocations of an abdominal viscus.

<sup>2</sup> Allbutt's "System of Med.," vol. iii., p. 592.

may be derived by wearing a well-fitting, adjustable abdominal belt. Flannel is the best material if the patient's skin tolerates it. The treatment of the neurasthenic symptoms and dyspeptic symptoms respectively is given elsewhere (Chapter X.). In severe cases which resist milder measures the idea of operative procedure should certainly be entertained. Cases have been recorded in which all the symptoms disappear after a simple abdominal incision, and the subsequent compulsory rest. In other cases definite organic lesions were found within the abdomen, which had not been detected during life, and which apparently caused the displacement.

§ 181. **Incipient or obscure visceral or spinal disease.** a. In cases of chronic pain GENERALISED OVER THE ABDOMEN one might suspect cancer of the intestines, of the pancreas, or of the kidney, cancer or tubercle of the suprarenals (*i.e.*, Addison's disease, in which pain over the stomach is a constant sign), or other incipient disorders, rheumatism of the abdominal muscles, enteroptosis, or movable kidney. Children may suffer from recurrent attacks of abdominal pain for which no cause can be found. Such cases, Dr. Guthrie says,<sup>1</sup> should be treated as incipient intussusception—that is to say, avoid purgatives and give digestible foods, and small doses of opium.

b. In various spinal affections the pain is frequently referred to the FRONT OF THE ABDOMEN, and among the more obscure causes may be mentioned abdominal aneurysm pressing on the spine, and cancer or caries of the vertebrae. The first of these occurs mostly in male adults, the second in the aged, and the third (Pott's disease) in children. In the latter the child frequently refers to the pain as "stomach-ache," worse after running about. The girdle pain of chronic and acute myelitis should also be borne in mind.

c. If the patient complain of PAIN SITUATED CHIEFLY IN THE LOWER ABDOMEN, one might suspect appendicitis (*vide supra*), cancer or other disorders of the bladder, tubercle or cancer of the prostate or testes, peri- and para-metritis (in which there is a good deal of pain shooting down the legs), extrauterine pregnancy, pyosalpinx, dysmenorrhœa and all its causes, uterine neuralgia, and obturator hernia. Hemorrhoids are sometimes attended by pain in the abdomen (which disappears upon the cure of these), and so also are new growths and various ulcers of the lower bowel. And, finally, among the unsuspected causes I have seen pelvic hydatid in a boy of 10.

d. PAIN SITUATED CHIEFLY IN THE UPPER ABDOMEN may be due to various affections of the liver, stomach, and spleen. Among the painful affections of the *liver*, perhaps perihepatitis and cancer are the commonest; hydatid is one of the obscure conditions, though it is rarely painful. Abscess above or below or within the liver should be suspected in those who have resided in tropical countries. Among the painful affections of the *stomach* may be mentioned gastric (or duodenal) ulcer, gastritis (acute or chronic), cancer of the stomach—which in its most usual form, scirrhus of the pylorus, is commonly very obscure in its early stages—and gastralgia. Painful affections of the *spleen* are not common, but the capsule is sometimes the seat of a painful inflammation.

1 "Treatment," May 12th, 1898.



§ 182. Diseases of the pancreas are fortunately rare, for they are always very obscure, and are seldom recognised during the life of the patient.

As far as our present means of investigation go, the *symptoms* to which they give rise are—(1) *abdominal pain, deep-seated in the epigastrium* radiating to the l. shoulder, and round the l. loin; (2) nausea, and vomiting of glairy mucus, anorexia, and acid eructations; (3) great debility, rapid emaciation, and mental depression; (4) undigested fat and muscle fibre in the feces; 1 and (5) when salol is administered by the mouth for 24 hours it does not appear in the urine as carbolic acid. As to *physical signs*, a tumour may be felt only when the disease is very advanced.

The diseases of the pancreas which have been recognised, chiefly after death, are as follows:—

I. HÆMORRHAGE WITHIN THE PANCREAS, a rare condition which, if of any extent, causes death in 24 hours or less (§ 172).

II. PANCREATIC CYSTS due to obstruction or obliteration of the duct by biliary or pancreatic calculi, or cicatricial contraction. Fatty diarrhœa does not occur in these cases. The fluid withdrawn by aspiration will emulsify fat, and convert starch into sugar.<sup>2</sup>

III. PANCREATIC CALCULI are small concretions consisting chiefly of carbonate of lime.

IV. ACUTE PANCREATITIS is according to Dr. R. H. Fitz<sup>3</sup> met with in 3 forms: 1. *Acute Hemorrhagic Pancreatitis*, which sets in suddenly with agonising pain, and results in death in 1 to 4 days (§ 172). 2. *Acute Suppurative Pancreatitis* begins suddenly with pain and irregular pyrexia, and may lead to death in 3 or 4 hours, but Fitz's cases more often became chronic, and lasted some months. There may be several small or one large abscess. 3. *Gangrenous Pancreatitis*, in which necrosis of the organ occurs, and it may be passed as a slough by the bowel. Two of Fitz's cases recovered.

V. CHRONIC PANCREATITIS is a fibrosis of the organ which mostly runs a latent course; but has received considerable attention of late years because it is frequently associated with diabetes, especially in those cases where atrophy of the gland ensues.

PANCREATIC DIABETES. The association of glycosuria with pancreatic calculus was first pointed out by Cowley in 1788. But it was Laucereaux in 1877 who maintained there was a special form of Diabetes dependent on grave alterations in the pancreas (Pancreatic Diabetes), characterised by polyuria, excessive thirst and appetite, rapid loss of flesh, and glycosuria. Pancreatic diabetes may, however, occur with lesions of the pancreas other than chronic pancreatitis; and grave alterations of the organ may exist without diabetes, the occurrence of which probably depends on loss or destruction of the gland cells.

VI. CANCER OF THE PANCREAS may be primary or secondary, but is a very rare condition. It is said to occur in about 6 per cent. of all cancers (Segre). The symptoms are: (1) Pain in the epigastrium, which at first occurs in paroxysms, then becomes constant and runs a chronic course. (2) Symptoms of gastric disorder may be present for months before any other symptom. (3) Jaundice, intense and persistent from pressure on the bile duct, is usually present, and sometimes pain like biliary colic accompanies this. (4) The other symptoms are those above described. (5) Later on, a tumour is found in the epigastrium or in the umbilical region, with little or no mobility, deep-seated and hard to define.

The *Diagnosis* of cancer and other tumours of the pancreas is always difficult. A tumour of the liver, pylorus, or transverse colon is more mobile. Much indican in the urine points to an intestinal rather than to a pancreatic tumour. No great stress can be laid on the presence of fat in the feces or on glycosuria, but abundant undigested muscle fibre found in the feces is more characteristic of pancreatic disease.

*Prognosis.* In cancer of the pancreas death usually occurs within 4 weeks after the onset of jaundice, or 6 weeks after ascites sets in. Emaciation and debility may not come on till late in the disease. The complications are: (i.) symptoms due to pressure on the neighbouring organs—intestine, stomach, or portal vein; (ii.) sudden hæmorrhage into the alimentary tract or the peritoneal cavity; (iii.) pulmonary embolism. Sudden death occurs in the last two.

*Treatment.* This is mainly symptomatic. The administration of pancreatin or similar preparations may aid the digestion.

<sup>1</sup> The presence of fat and muscle fibre in the feces has not the same pathognomonic value formerly attributed to it. Their occurrence depends on the amount of gland tissue which is diseased.

<sup>2</sup> A case of retroperitoneal rupture of a pancreatic cyst occurring in a young man about 25 years of age was admitted in the Paddington Infirmary with all the symptoms of acute peritonitis. Laparotomy was performed by Sir Fredk. Treves, but nothing was found until after death, 48 hours later. The origin of the cyst was not even then discovered, but the cellular tissue behind the peritoneum was infiltrated with the usual putaceous material.

Middleton-Goldsmith Lecture, *New York Medical Record*, 1869, vol. i.

## GENERALISED ABDOMINAL ENLARGEMENT.

§ 183. **Classification.** Generalised abdominal enlargement occurs under 4 conditions—

- I. Solid abdominal tumours . . . . . § 189
- II. Gas in the intestines (tympanites), or occasionally in the peritoneum . . . . . § 184
- III. Fluid in the peritoneum (ascites) . . . . . § 186
- IV. A cystic collection of fluid in the abdomen . . . . . § 187

The **routine procedure**, as previously described (§ 168), should be—by Inspection, Palpation, Percussion, Mensuration.

If a **hard tumour** can be felt in any part, turn first to § 189.

If the abdomen is quite **soft to palpation** and **resonant** all over, turn first to § 184.

If the abdomen is **dull to percussion** in the flanks, and presents the fluctuation test, turn first to § 186.

If the abdomen is **resonant in the flanks** and **dull in front**, turn first to § 187.

*The abdomen is uniformly enlarged; it is soft and yielding to palpation; and percussion, systematically conducted over the whole area, gives a RESONANT note—the swelling is probably due to TYMPANITES.*

§ 184. **Tympanites** is the term employed for a flatulent distension of the stomach and intestines by gas. It should be remembered that flatulent distension may accompany and render obscure a *small quantity of fluid in the peritoneum*.

The *causes* of tympanitic enlargement are as follows:—

I. Atonic and other forms of **DYSPEPSIA** are the most frequent causes of flatulent abdominal distension. It is usually intermittent and is generally greatest after meals (§ 205).

II. In **ATONY OF THE COLON** the bowels are constipated, and the patient, an adult, is liable to “colicky” pains; but there are no constitutional symptoms or emaciation as in the next cause.

III. In **TUBERCULAR PERITONITIS** there is a tendency to the formation of intestinal adhesions and *flatulent distension*. In tubercular peritonitis, moreover, the distended abdomen has a doughy feel, and here and there a dulness on percussion which is

quite characteristic. It is accompanied by emaciation and an evening rise of temperature.

IV. "PHANTOM TUMOUR" may assume the shape of a generalised more or less resonant enlargement; but it more often resembles a localised tumour (§ 189).

V. In OBSTRUCTION OF THE BOWELS there is considerable abdominal distension accompanied by pain, vomiting, and other general constitutional disturbance (§§ 230 and 231).

**Gas in the peritoneum** gives much the same signs as tympanites; only there is extreme distension, and hyper-resonance all over to such a degree that the normal dullness of the liver and spleen is obscured. It is met with only when perforation of some part of the alimentary canal occurs. The patient is collapsed, and presents all the symptoms associated with perforation (§ 170). A few hours after the occurrence of the perforation, a delusive lull occurs in the collapse and other symptoms, only to be succeeded by a fatal exacerbation. Perforation of gastric ulcer is the commonest cause, and one of the diagnostic features of this condition is the loss of the normal area of liver dullness.

*There is uniform abdominal enlargement, which is soft and yielding to palpation and DULL TO PERCUSSION in parts; the FLUCTUATION SIGN is present—there is FLUID WITHIN THE ABDOMEN.*

§ 185. When there is **fluid in the peritoneum**, either free or encysted, the belly is soft to palpation, dull to percussion in parts (either in the flanks or in front); and the measurements show the abdomen to be uniformly enlarged.

When the fluid is in any quantity, two special signs can be elicited. 1. *Fluctuation test.* When a large amount of fluid is present, a wave of *fluctuation* may be seen to travel across the surface when we tap or "flip" one side. This can only be satisfactorily elicited when the abdomen is full and tense. 2. *Percussion test.* A percussion wave can be transmitted from one hand to the other *through the fluid*, by the law that fluids transmit pressure, or a blow, equally in all directions. Place the left hand over one side of the dull portion and tap sharply with the fingers of the r. hand over the opposite side; an impulse

will be felt by the l. hand if fluid be present. Neither of these signs can be elicited in a gaseous enlargement or a solid tumour.

But the fluid may be either, (a) FREE in the peritoneal cavity, when it is termed ascites, or (b) enclosed in a CYST, such for instance as an ovarian cyst.

(a) If FREE in the peritoneal cavity, it will obey the law of all fluids and *shift with the position of the patient*. Thus in Ascites (§ 186), when the patient is on his back you will find both flanks are dull to percussion and the umbilical region is resonant; then, if the patient turns on to one side you will find that the uppermost flank which was before dull is now resonant, and the umbilical region, if there is much fluid, is dull. Much may be learned from the character of the fluid withdrawn by a trochar. Ascitic fluid is straw-coloured, with much albumen. Hæmorrhagic fluid usually means cancer.

(b) If the fluid is ENCYSTED, *e.g.*, Ovarian cyst, we can still elicit the fluctuation and the percussion tests just referred to, but the level of the dulness will not alter with the position of the patient (§ 187).

*Fallacies of these tests for fluid.* In obese persons considerable difficulty arises in the detection of fluid, because the fluctuation test can be elicited whether fluid be present or not. In applying the "percussion test" for fluid in obese subjects, an assistant should place the edge of his hand vertically on the umbilicus. This will prevent the wave or impulse from travelling across the surface of the omental and subcutaneous fat, instead of through the fluid.

*There is a generalised uniform enlargement of the abdomen which gives all the SIGNS OF FLUID, and the fluid ALTERS ITS LEVEL with the position of the patient*—the disease is ASCITES.

§ 186. **Ascites** is a term applied to an effusion of non-inflammatory fluid within the peritoneum (dropsy of the peritoneum). The physical signs of fluid have just now been described above. It is sometimes difficult to detect a very small quantity of fluid in the peritoneum, but its existence is rendered probable (i.) by the dulness on percussion of the umbilical region with the patient on his hands and knees, and (ii.) by finding that when the patient turns on to one side the flank which was dull is now resonant.



Ascites may have to be *diagnosed* from any of the cystic conditions mentioned below (§ 187), but certainly the most frequent and important source of difficulty is *ovarian cyst*. In ascites (i.) the flanks bulge, (ii.) the front is flat and resonant, and (iii.) both flanks are dull, but if the patient turns on his side the upper flank becomes resonant—three features which are the exact reverse of those found with ovarian and other cystic tumours. See also Table on p. 329. Occasionally peritoneal adhesions (especially cancerous) may confine the fluid to one part of the abdomen, and then the fluid does not shift with the position of the patient. A greatly distended urinary bladder may simulate ascites, but the passage of a catheter readily excludes this fallacy.

The other *symptoms* which accompany ascites belong to two categories. (1) Those due to pressure within the abdomen—*e.g.*, œdema of the feet and legs, from pressure on the vena cava and its branches; later on dilatation of the surface veins of the anterior abdominal wall may occur from the same cause; albuminuria from pressure on the renal veins; and dyspnœa from mechanical impediment in the circulation. (2) There are evidences of the condition which has caused the ascites; and of all the causes by far the commonest is alcoholic cirrhosis of the liver. The temperature is generally normal, excepting in cause IV, below.

The *causes of ascites* are five in number. In reference to the diagnosis of these causes, if there be any œdema of the ankles, it is important to ascertain whether this œdema or the ascites came first. For instance, when PORTAL OBSTRUCTION is in operation, the dropsy of the feet will have started subsequently to the ascites; in HEART or LUNG disease it will have preceded the ascites; whereas in RENAL DISEASE they would have started about the same time. ASCITES with well-marked JAUNDICE in an old person is extremely likely to mean CANCER OF THE LIVER or peritoneum. ASCITES with SALLOWNESS of the skin in a MIDDLE-AGED person is most probably ALCOHOLIC CIRRHOSIS of the liver.

I. **Portal obstruction** is the commonest cause of well-marked ascites. The fact that this cause is in operation is recognised in two ways—(a) by a history or presence of the *symptoms* of

portal obstruction (of which ascites is only one); and (b) the presence or a history of one of the *causes* of portal obstruction.

(a) The SYMPTOMS of portal obstruction, in the order in which they usually appear, are as follows: (1) A liability to attacks of gastric and intestinal catarrh, as evidenced by pain in the stomach, irritable dyspepsia, alternating diarrhœa and constipation, and the vomiting of mucus streaked with blood, especially in the early morning before breakfast. (2) Hæmorrhoids. (3) Hæmorrhage, sometimes in very large quantity, from the stomach and the bowels. (4) Congestion, and therefore enlargement of the spleen. (5) ASCITES is one of the later results. (6) Enlargement of the veins of the abdominal wall from the establishment of a collateral circulation. (7) Edema of the legs also appears subsequently to the ascites, and is due to pressure on the large veins in the abdominal cavity by the ascitic fluid. (8) Albumen in the urine may arise in the same way, or from concurrent disease of the kidney; in the former case the albuminuria may disappear after paracentesis.

(b) The CAUSES of portal obstruction may be grouped into (a) diseases within the liver, or ( $\beta$ ) diseases outside it.

(a) *Diseases within the liver.* *Cirrhosis* of the liver is by far the commonest of all the causes, and this is nearly always due to alcoholism, there being a history of this and alcoholic dyspepsia. Simple ascites without marked jaundice or other obvious symptoms is presumptive of cirrhosis. *Cancer* produces portal obstruction usually by the pressure of the enlarged glands in the fissure or by masses protruding outside the liver. *Perihepatitis* sometimes produces ascites by puckering of the capsule. Ascites only very rarely accompanies *hepatic congestion*, and never fatty liver, hydatid, or abscess.

( $\beta$ ) The causes of portal obstruction *outside the liver* are: (1) *Cancer* of the stomach, duodenum, or pancreas, and various other tumours pressing on the vein. (2) Enlargement of the *glands* in the fissure of the liver (cancerous, tubercular, or syphilitic). (3) *Thrombosis* of the portal vein is rare, and only arises when some general cause for thrombosis is in operation. Here the symptoms are very acute.

II. In **heart disease**, either primary (*e.g.*, mitral disease and cardiac dilatation) or secondary to lung mischief, the ascites is

generally part of the dropsy affecting the cellular tissues and other serous cavities of the body. Here dropsy of the *feet will have preceded the abdominal dropsy*, and there will be previous history of palpitation, dyspnœa, and perhaps cough. An examination of the heart will also reveal the nature of the disease.

III. In **kidney disease** ascites may be part of a General Dropsy affecting the face, limbs, peritoneum, pleuræ, and pericardium. The fact that the dropsy started in all of these situations about the same time reveals this cause. Albuminuria is frequently enough a consequence of the pressure of the ascitic fluid, but the presence of epithelial casts almost certainly indicates that the renal disease was primary. It usually takes the form of acute or chronic parenchymatous nephritis, very rarely waxy or granular kidney.

IV. **Chronic peritonitis** is another cause of fluid in the peritoneum. An idiopathic form of chronic peritonitis is sometimes described, but it is practically never met with apart from a deposit of tubercle (in the YOUNG) or of cancer (in the AGED) (§ 177). In the tubercular form adhesions rather than fluid are met with; in the cancerous it is *vice versa*.

V. A small amount of effusion into the peritoneum is found in severe **anæmia**, especially pernicious anæmia, and some other blood disorders; but it is never very great.

The *prognosis and treatment of ascites* is very largely that of the morbid condition with which it is causally related; and the reader should turn to cardiac disease, renal disease, or chronic peritonitis according to the case. Again, the *prognosis of ascites due to portal obstruction* depends very much on the nature of the intra- or extra-hepatic lesion which has produced it, as given above and in Chapter XII. The degree of the obstruction is measured by the amount of ascites and other symptoms present; and still better by the amount and frequency of the hæmorrhage that has taken place from the stomach or intestines. Life may be prolonged for many years, even when a considerable amount of ascites has accrued, provided it has come on slowly, and time has thus been afforded for the gradual establishment of the collateral circulation through the surface veins of the abdomen and other collateral channels. It is in this sense, that repeated tapplings are good,

for in this way time is gained for the establishment of collateral circulation. In cases of alcoholic cirrhosis the habit must be abandoned, otherwise the patient cannot live longer than six to twelve months, for ascites indicates an advanced condition of cirrhosis.

The *treatment of ascites*, like its prognosis, must depend upon its cause (*q.v.*). The treatment of *ascites due to portal obstruction*, and to some extent that of other forms, is as follows: (1) Hydragogue purgatives are certainly called for, and mag. sulph. and the other salines are the best. Elaterium seems particularly valuable if given in sufficient quantities to produce three or four watery stools a day. (2) Diuretics are recommended by some, but, in my experience, there is no form of dropsy in which they are of so little use as in ascites, at any rate until the pressure has been relieved by tapping. Copaiba resin, and cubebs are sometimes useful, and I have in one or two cases given pil. digitalis co. with some benefit after repeated tapping. (3) Tonics are useful combined with the preceding, such as a mixture containing pot. bitart., fer. tart., and digitalis. (4) Paracentesis is generally called for sooner or later. Some physicians say it should be put off until it is called for by the urgency of dyspnoea. In cancer this is certainly a good rule, but in cirrhosis of the liver it is best to operate at once in all cases where there is much fluid, unrelieved by medicine. It is often found that medicines which were useless before are efficacious after the operation, because the kidneys are relieved from pressure. Sometimes complete recovery takes place after repeated paracentesis, because time is thus afforded for the establishment of the collateral circulation as above mentioned. It is best to use a small trochar with the tube conducted to the pail, so that the peritoneum may gradually empty itself. With a large one leakage may remain, or peritonitis may ensue. In 1896 a surgical method of promoting the collateral circulation by the artificial production of peritoneal adhesions in cases of alcoholic cirrhosis was introduced by Dr. D. Drummond and Mr. J. R. Morison<sup>1</sup> which has been attended by a measure of success.

<sup>1</sup> *Brit. Med. Journ.*, vol. ii., 1896. See also a paper by Rolleston and Turner, *The Lancet*, Dec. 16, 1899.



There is a **generalised abdominal enlargement** which gives all the signs of fluid (§ 185); but the fluid does NOT ALTER ITS LEVEL with the position of the patient—there is ENCYSTED FLUID (probably ovarian) IN THE ABDOMEN.

By far the commonest of such cystic tumours is an OVARIAN CYST. Other and less common cystic abdominal tumours are HYDRAMNIO, CYSTIC FIBROMA of the uterus, HYDRO- and PYONEPHROSIS, PANCREATIC CYST, a large HYDATID, a cyst of the GALL-BLADDER, and ENCYSTED ASCITES.

§ 187. I. **Ovarian cyst**<sup>1</sup> is centrally situated, and grows from below upwards. The enlargement is fairly uniform, and it gives all the signs of fluid (§ 185). But the level does not alter with the position of the patient; and whereas the umbilical region is dull on percussion, the flanks are resonant. On palpation it is tense and elastic; and in malignant ovarian cysts nodules can be felt in the walls. The diagnostic features between ascites and ovarian cysts are given in Table XVI.

TABLE XVI.

	ASCITES.	OVARIAN CYST.
<i>Inspection.</i>	Flanks bulge, front flat.	Flanks flat, front bulges.
<i>Percussion.</i>	Flanks dull, front resonant. On turning, upper flank becomes resonant.	Flanks resonant, front dull. No alteration of dullness on turning.
<i>Measurement.</i>	Umbilicus to xiphoid greater than umbilicus to pubes. Circumference at umbilicus greater than slightly below. Navel to iliac spine same both sides.	Umbilicus to xiphoid less than umbilicus to pubes. Circumference at umbilicus less than slightly below. Navel to iliac spine greater one side than another.

The associated *symptoms* with it are—(1) a history of it having grown upwards from the pelvis, and (2) these tumours (unlike ascites) may be of very rapid growth, and reach quite a large size in three or four months. (3) There have usually been menstrual irregularities, though by no means always. There may have been no general symptoms of any kind, but generally some pain and local discomfort have been complained of. Often when the cyst contains pus there is little or no fever. When there is a history of attacks of pain, it generally indicates adhesions, an

<sup>1</sup> Parovarian cysts are rare. They present much the same symptoms as ovarian cysts.

important matter from an operator's point of view. An examination of the uterus usually reveals nothing. A malignant cystic ovarian growth is indicated by (1) the presence of nodules in the walls; and (2) the age of the patient and a history of emaciation, and severe pain.

*Diagnosis.* The characters of the fluid withdrawn by aspiration from the several abdominal cysts are given in a later chapter.

In the *earlier stages* the diagnosis of an ovarian tumour is sometimes difficult. It is an elastic, movable, and globular swelling; the uterus is not enlarged, and it can be defined as quite separate from the tumour. In this stage it may have to be diagnosed from *hydro- or pyo-salpinx*. *Para- and peri-metritic* exudation and *pelvic hæmatocele* would be very firmly fixed in the pelvic cavity and accompanied by constitutional symptoms. In *extra-uterine foetation* there would be morning sickness, a patulous os uteri, and other symptoms of pregnancy, with an empty uterus. These are all dealt with in Chapter XIV.

In the *later stages* ovarian cysts have to be diagnosed from all the conditions mentioned below.

II. PREGNANCY WITH HYDRAMNIOS and a thin uterine wall is sometimes very difficult to diagnose from an ovarian cyst, for both develop very rapidly. Experienced clinicians have been known to fail in the differentiation. The symptoms of pregnancy (see § 329), the exactly central position of the tumour, and, as a last resort, the careful use of uterine sound, may aid us in the diagnosis. *Hydatid mole* presents similar difficulties, but it is fortunately more rare.

III. LARGE CYSTIC FIBROMA of the uterus, especially of the sub-peritoneal variety, may produce the signs of a fluid tumour. It is recognised by (1) its connection with the uterus, which is enlarged; and (2) its slow growth, which may extend over many years; and (3) *menorrhagia* in some cases.

IV. A LARGE HYDATID CYST of the spleen or liver, a HYDRO- or PYONEPHROSIS, a dilated GALL-BLADDER, a large PANCREATIC or OMENTAL CYST, or a large PERITYPHLITIS ABSCESS, may on rare occasions produce the appearance of a general fluid enlargement of the abdomen, and may require to be diagnosed from ovarian dropsy: but they are nearly always *asymmetrical*. They grow from, and their percussion dullness is continuous with, the organs whence they rise: they are referred to among Abdominal Tumours (§ 188). Doubtful cases can generally be cleared up by withdrawing and examining a small quantity of the fluid by means of an aspirator: the characters of the fluid are given in a later chapter.

V. ENCYSTED ASCITES is not common. It may result from previous peritonitis, of which there will probably be a history. More frequently, perhaps, it results from tubercle or cancer of the peritoneum (§ 177). In very rare cases congenital deficiency or adhesions may exist. In all of

these there is a want of symmetry in the enlargement and in the fluid, an absence of the associated symptoms of ovarian tumour, and a history or other evidences of the cause in operation.

The *prognosis* of ovarian tumour is always serious, though in the non-malignant form it may be quiescent for some years. If not treated, a cyst may go on (1) to rupture and fatal peritonitis ; (2) it may become inflamed ; (3) the pedicle may become twisted ; (4) hæmorrhage may take place into its cavity.

The *treatment* is entirely surgical, and the earlier it is removed the better, and before the attacks of pain indicating inflammatory adhesions take place. Tapping is a temporary measure only, it certainly increases the risks for future operation, and is only justifiable for diagnostic purposes.

#### ABDOMINAL TUMOURS.

§ 188. **Method of procedure.** We now turn to the second group of abdominal enlargements, namely, those in which the enlargement has originated in or is localised to one part, *i.e.*, Abdominal Tumours. It is only by repeated and careful examination that mistakes can be avoided in the diagnosis of abdominal tumours. The same methods are adopted here as in general enlargement (§ 168), which should be consulted. (1) *Inspection* should never be omitted in the recumbent, and sometimes in the erect, posture ; (2) *percussion*, to define the boundaries and nature of the tumour, and its continuity with some organ ; (3) *palpation*, with a flat hand previously warmed and with the abdominal muscles thoroughly relaxed by a suitable posture ; and (4) careful *measurement* made and recorded both for comparison one part with another, and to see what progress the growth makes.

*Fallacies of abdominal tumours.* (1) *Fat in the omentum* (obesity) may offer a serious obstacle to the examination of abdominal enlargements or tumours. The only way in these circumstances to arrive approximately at a correct decision is to place the hand flat upon the belly and then dip the fingers suddenly and forcibly inwards. The stomach and intestines should be empty.

(2) The presence of *fluid* within the abdomen, as well as a solid tumour, may prevent our discovering or examining it thoroughly. The difficulty may be obviated to some extent by suddenly flexing the fingers as in the case of obesity.

(3) *Fæcal accumulations* may simulate malignant and other tumours, though they can generally be indented by the fingers. They are always situated in some part of the large bowel. In doubtful circumstances a course of castor oil or other hydragogue purgative is desirable. But they may exist for many weeks in spite of purgatives.

(4) A "*phantom tumour*" is a swelling (usually tympanitic, sometimes dull), produced by irregular muscular contraction, and it is wonderful how precisely it may simulate a solid tumour. It is apt to appear and disappear suddenly, hence the name. The condition is met with for the most part in young hysterical women, and is quite beyond the control of the patient. It is a frequent cause of error in diagnosis. It is generally due to spasmodic contraction of one or both recti muscles. Spasms of the diaphragm may produce a generalised abdominal enlargement by pushing the viscera down. The patient should be placed in a position of perfect ease for the relaxation of all the muscles of the body, with the knees drawn up and the neck slightly bent. Sometimes nothing but the administration of an anæsthetic to complete narcosis will enable us to establish the diagnosis; and this must be done in cases of importance.

Having excluded these fallacies, and being satisfied as to the existence of an abdominal tumour, there are five points to which our attention should be directed:—

1. The first and most important question is the *locality of the tumour*; what region is it situated in, or where did it start?

2. To ascertain with *which organ it is connected*, consider what organs are located in the region occupied by the tumour, and then see if it be structurally continuous by palpation and percussion with one of these.

3. If it *moves with the breathing* of the patient, we know that it must be connected with the diaphragm, or some organ depressed by it during respiration, such as the spleen, liver, stomach, intestines, or omentum. If fixed, it is a tumour of the kidney (unless it be dislocated), aorta, lymphatic glands, or some other organ unaffected by respiration, or bound down by adhesions.

4. Inquire for a *history of any disease* or functional disturbance of the abdominal organs—*e.g.*, in case of the kidney, whether the



urine contains, or has contained, blood or pus, although the tumour may appear to be far from these organs; or perhaps there has been jaundice pointing to hepatic mischief.

5. The diagnosis of the *nature of the tumour* depends very largely upon its history and the age and sex of the patient. Tense cystic tumours are extremely difficult to differentiate from solid growths, but we can try to obtain the percussion and fluctuation tests (§ 185, *ante*). In obscure cases the use of a long thin exploratory trochar is justifiable; and then the presence of the opalescent fluid of hydatid, the blood-stained fluid of malignant disease, the straw-coloured fluid of encysted ascites, a fluid which will digest starch and emulsify fat (pancreatic cyst), or the absence of any kind of fluid, will materially help us to arrive at the precise nature of any given tumour. There is also another question which very frequently presents itself for consideration—viz., is the tumour benign or malignant? The general symptoms of malignant disease (cancer) are discussed under Emaciation with Debility, Chapter XVI.; but the age of the patient, and the rapid course and lethal tendencies of the disease, are the chief means of differentiating it.

§ 189. *If there is a visible or palpable tumour in the abdomen, ascertain which REGION the tumour chiefly OCCUPIES or ORIGINATED in, and refer to that region in the following summary. Having identified ITS ORIGIN in this way, reference must be made to the diseases of the organ affected to ascertain the NATURE of the tumour.*

I. RIGHT HYPOCHONDRIUM. The commonest tumours in this position are tumours of the *liver*, especially cancer and enlargement of the organ. The features which HEPATIC TUMOURS present in common, in addition to their position, are: (1) They are not covered in front by resonant bowel, and their dulness is continuous with that of the liver; (2) they move with respiration; and (3) there are ascites, jaundice, and other evidences of liver derangement. It must not be forgotten that hepatic tumours may be simulated when the liver is pushed down by emphysema, by pleuritic and pericardial effusions; or that it may be puckered by contraction of the capsule, and so simulate a tumour or enlargement (Diagnosis of hepatic enlargements, § 237); Riedel's lobe (see

below) is another fallacy. Dilatation of the GALL-BLADDER (*e.g.*, by gall-stones) is recognisable as a tense rounded swelling below the ninth costal cartilage. There is only occasionally a history of biliary colic, but always a history of "chills" (biliary fever), see § 243. Tumours in this region may also be connected with the *duodenum* or *right kidney* (see II. and IV. below).

*Riedel's Lobe*<sup>1</sup> (lingiform or floating lobe of the liver). In certain cases, usually associated with gall stones retained within the gall-bladder, a tongue-shaped process projects downwards from the r. lobe of the liver, or the lobus quadratus. It may reach as far as the iliac crest or even to the iliac fossa. In hardly any of the cases in which it has been observed (Glénard<sup>2</sup> collected 80) has the condition been correctly diagnosed until operation or an autopsy was performed. It has most often been mistaken for floating kidney; and has also been taken for distended gall-bladder, hydatid cyst, new growth, and omental tumour. It is sometimes tender, its shape more or less that of a pear. Under chloroform its connection with the liver might possibly be made out.

*Suprarenal tumours* become manifest in the right or left hypochondrium, and are difficult to distinguish from tumours of the liver, gall-bladder, and spleen respectively. Mayo Robson<sup>3</sup> summarises the symptoms thus: (i.) pain radiating from the tumour across the abdomen and to the back; (ii.) pain complained of at the shoulder tip; (iii.) emaciation, with nervous depression, and digestive disturbance; (iv.) a tumour felt beneath the costal margin (right or left), at first movable with respiration, but soon fixed; and it can be felt posteriorly in the costo-vertebral angle; (v.) absence of urinary and gall-bladder symptoms.

II. In the *EPIGASTRIC REGION* tumours may be connected with the liver (*vide supra*); but the first tumour which would occur to one's mind would be *CANCER OF THE STOMACH*—*i.e.*, a hard irregular swelling attended by vomiting, "coffee-ground" in character. The commonest form of malignant disease of the stomach, however, is *scirrhus* of the pylorus, and in this copious vomiting at long intervals and other gastric symptoms appear long before any swelling can be detected (§ 209). *Tumours of the duodenum* may sometimes be distinguished from those of the stomach by their immobility during a deep respiration.

*Pancreatic cysts* may cause a fluctuating swelling in the epigastrium, but their detection is extremely difficult. There may be a history of pain<sup>4</sup> and symptoms of pancreatic disease (see § 182). Cysts of the *small omental sac* present a similar swelling. *Pulsation in the epigastrium* is a symptom of that frequent condition, dilated r. ventricle, and it must not be mistaken for abdominal aneurysm (*vide infra*).

<sup>1</sup> "Berlinen Klinische Wochenschrift," 1888, Nos. 29 and 30.

<sup>2</sup> "Les Ptoques Viscérales," Paris, 1899.

<sup>3</sup> *Brit. Med. Journ.*, ii., 1899, p. 1100.

<sup>4</sup> Case recorded by A. M'Phedran, *Brit. Med. Journ.*, i., 1897, p. 1400.

III. In the LEFT HYPOCHONDRIUM tumours of the SPLEEN originate, and sometimes they attain to an enormous size. These are fully discussed in § 263. They move with respiration; and they make their way forward in *front* of the colon. The tumour can generally be moved forwards by getting the hand behind it; a procedure which distinguishes them from tumours of the left kidney. They resemble tumours of the left lobe of the liver, but these latter cannot be displaced downwards by the hand. Other tumours in this position may be connected with the *stomach, pancreas, liver, kidney, and sigmoid flexure.*

IV. The LUMBAR REGION may be the starting place for RENAL TUMOURS which are characterised by four features—(i.) Their fixity during respiration. (ii.) Dulness in one flank, and, unless both kidneys are involved, resonance in the other. (iii.) They are *always resonant in front* because as they make their way forward they push the colon in front of them; and (iv.) there is no resonant part between the dulness of a renal tumour and the spine, as there would be in the case of a splenic tumour. They generally retain their rounded and reniform shape. They are distinguished from hepatic tumours by the dulness in the flank not being continuous with that of the liver; and by the presence or history of blood, pus, or other urinary changes. The commoner forms of renal tumours are, hydro- and pyo-nephrosis, renal sarcoma (commonest tumour in children), and perinephric abscess. *Pyo- or Hydro-nephrosis* are cystic tumours, containing urine with or without pus respectively (see § 316). Hydronephrosis may be almost painless, not tender, and unattended by any subjective or constitutional symptoms; pyo-nephrosis is always tender, and attended by hectic fever and malaise. Other tumours starting in the lumbar regions may be connected with the *ascending and descending colon.*

*Movable kidney* is one of the most frequent of abdominal tumours. It may be found in any part of the cavity below the liver. Its mobility, rounded or reniform shape, are characteristic, but not always easily detected. There is a characteristic pain of a dull, aching, or dragging character in the back, increased by exertion (see § 178).

V. The LEFT INGUINAL REGION may be the seat of a tumour

caused by CANCER of the SIGMOID FLEXURE ; and this is the most frequent position in the bowel for cancerous deposit. Cancer and other *tumours of the large intestines* are distinguished generally by their free mobility (unless fixed by adhesions). They are, when cancerous (far the commonest neoplasm of the intestines), usually multiple, and attended by more or less ascites, and by some irregularity of the bowels, generally chronic diarrhœa. The commonest starting point for primary cancer of the bowel is, as just mentioned, the sigmoid flexure ; but before a cancerous swelling can be detected in the left inguinal region, the patient will have been troubled with recurrent diarrhœa and sciatica, sometimes melæna. These symptoms are followed in course of time by œdema of the leg. In the condition known as *colloid cancer* of the peritoneum all the intestines become matted together, and although fluctuation may be detected, there is little or no fluid in the peritoneal cavity. Cancer of the *small intestines* gives rise to hard, irregular, nodular, usually multiple tumours, and, in addition to the signs just mentioned, there are the age of the patient and cachexia. Constipation, going on sometimes to obstruction, may also be present. The prognosis of cancer is given elsewhere (Chapter XVI.). But so-called “colloid cancer” of the peritoneum is a remarkable exception in regard to its duration, and it may go on for many years before death occurs. The treatment, which is not very hopeful, is referred to under “Emaciation.”

VI. The RIGHT ILIAC REGION is the position in which APPENDICITIS is usually manifested; it is fully described under “Abdominal Pain” (§ 176). *Intussusception* of the bowel, which occurs mostly in childhood, gives rise to a soft sausage-shaped swelling generally situated in this region (see Intestinal Obstruction). *Pelvic cellulitis* may form a firm swelling in either iliac region. Its other features are (i.) vaginal examination reveals a tender swelling in the corresponding fornix, pushing the uterus to the opposite side ; (ii.) there is a history of acute pain and fever at the onset of the condition, frequently following childbirth or abortion. *Cancer of the cæcum*,<sup>1</sup> contrary to what we might expect, often constitutes a *movable* tumour in the iliac region, and is very apt to be mistaken for masses of fæces. Epithelioma of the cæcum

<sup>1</sup> Clin. Soc. Trans., November 24th, 1899.



may be attended by suppuration, so giving rise to abscess in this region with pyrexia. The history of such cases may run a long course, and excepting in the age of the patient, resemble chronic appendicitis. *Psoas abscess* points in this region; if originating from dorsal caries it presents below, if from lumbar caries above Poupart's ligament.

VII. The UMBILICAL REGION is the starting place of tumours connected with the pancreas, duodenum, mesenteric glands, and aorta, all of which are *immobile during respiration*; though a tumour in this position is far more often connected with the stomach, liver, or large bowel, which *move with respiration*. Enlargement of the *mesenteric glands* may be sometimes detected in spare subjects by grasping the two sides of the abdomen either between the two hands or the finger and thumb of one hand. When large enough to form a tumour they are fixed and matted together.

*Aneurysm of the abdominal aorta* is a pulsatile and expansile swelling also immobile during respiration. In thin subjects a thrill may be felt, and a murmur heard. In auscultating the abdominal aorta we must be careful not to produce a murmur by pressure of the stethoscope. It is attended always by a severe fixed neuralgic pain in the spine, and sooner or later breathlessness and cardiac signs. It is these latter symptoms which distinguish true aneurysm from "pulsatile aorta" (see below), and from a swelling in front of the vessel to which the pulsation has been communicated. An endeavour should be made to grasp the swelling on each side so as to observe the expansile nature of the tumour.

*Pulsating abdominal aorta*—throbbing in the belly. Dyspeptic subjects and nervous females are often troubled with marked pulsation of the abdominal aorta which is sometimes obvious both to the patient and the doctor. There is in this affection great local discomfort and even pain, with marked pulsation obvious to both inspection and palpation. The diagnosis from aneurysm rests partly on the fact that the pulsation is not limited to any part of the aorta, and partly that such rapid and violent action of the heart is not common in aneurysm.

VIII. The HYPOGASTRIC REGION is the situation whence BLADDER, UTERINE, and OVARIAN TUMOURS grow. *Ovarian tumours* (which are nearly always cystic) are usually characterised in the *early stages* by their free mobility unless they are malignant, and their rapid growth (§ 187). *Tumours of the bladder* are usually rendered sufficiently obvious by changes in the urine. *Tumours of the uterus* are similarly revealed by uterine symptoms, excepting perhaps some sub-peritoneal fibroids. These may reach a large size without any symptoms at all; their origin and relations are

readily detected by bimanual examination. *Pregnancy* causes a symmetrical enlargement, starting from the hypogastric region about the third month of gestation (§ 329). Among the rarer tumours in this region pelvic hydatid and pelvic hæmatocele may be mentioned.

The NATURE, PROGNOSIS, and TREATMENT of these various abdominal tumours are discussed under the organ with which they are connected.

§ 190. **Flattening or recession of the abdomen** is not a sign of any great importance. “*Ventre plat, enfant il y a*” is a French expression of the fact that the abdominal wall slightly recedes during the first two or three months of pregnancy. It is also met with in abstinence from food, and in wasting disorders such as cancer and tubercle. It may be present also in intestinal, hepatic, and renal colic, and it may occur as a consequence of excessive purging or vomiting. A hollow or “boat-shaped” abdomen is said to be characteristic of meningitis in infants.

## CHAPTER X.

### THE STOMACH.

Two features cannot fail to strike the student in this department of medicine. The first is that we are very largely dependent upon subjective symptoms in the investigation of disorders of the stomach, a large proportion of the disorders of this organ being functional; and until the use of the "test-meal" (§ 200), and methods for estimating the motor-power of the stomach (§ 199), were adopted, we had to rely almost entirely upon the patient's sensations before and after meals to know how the stomach had been discharging its functions. The other feature relates to the important and widespread effects which derangements of the stomach produce in the general economy. The nutrition of course fails; but, apart from this, sufferers from gastric disorders are always liable to depression, which may sometimes be extreme. Prostration is apt to occur in all acute abdominal diseases; but in chronic disorders of the stomach the functions of the nervous system may be so profoundly disturbed by neurasthenic and other symptoms that the physician may overlook the primary cause of the mischief, namely, malassimilation of food.

#### *PART A. SYMPTOMATOLOGY.*

The symptoms which reveal disorders of the stomach may be **local** (viz., epigastric pain or discomfort, nausea or vomiting, hæmatemesis, dryness or bad taste in the mouth, flatulence, heartburn, water-brash, thirst, altered appetite); or **general** and **remote** (viz., cardiac symptoms, various nervous derangements, skin symptoms, and emaciation).

Among the **Local Symptoms** of gastric disorder, PAIN or DISCOMFORT AFTER FOOD, and NAUSEA or VOMITING, are perhaps the most constant and important, *i.e.*, the cardinal symptoms. HÆMATEMESIS is less frequent, but more serious. The other local symptoms are also of much value for diagnostic purposes.

§ 191. **Gastric pain**, or discomfort, in diseases of the stomach, is a most important *local* feature. Although it is not in every case sufficiently constant in its characters to enable us to establish the diagnosis, nevertheless it merits the closest study. In some cases it is altogether absent (even when simple ulcer or malignant disease exists), but when present, the features which should be noted are : its *position*, its *character*, its *degree*, its *constancy*, and above all, its *relation to the taking of food*.

Its *position* is usually over the epigastrium, but pain is very frequently complained of between the shoulders, and very severe pain in the back may also occur. In its *character* it varies considerably. Sometimes it is like a dull weight or a feeling of distension, such as occurs in atonic dyspepsia and chronic gastritis ; or it may be of a burning character, and such is the pain of acid dyspepsia ; or it may resemble abdominal cramp, as in spasm of the pylorus, or in some cases of gastralgia. Sharp or lancinating pain of a continuous character usually attends ulcer or cancer of the stomach.

Its *relation to food* is by far the most important feature of the pain in gastric diseases :—(a) *It comes on at once* and lasts a variable time in atonic dyspepsia, in acute gastritis, and in ulcer (simple or malignant). In simple ulcer the pain is at once relieved by vomiting, a very characteristic feature. (b) When pain *comes on an hour or more after food*, it is due to excessive acidity, either from hyper-secretion or fermentation (organic acids), and this again is very typical. In hyper-secretion, pain is relieved by taking food, but not in fermentation. A sudden attack of excruciating pain coming on in the middle of the night is characteristic of permanent hyper-secretion. (c) Pain coming on at a *late period of digestion* is characteristic of decomposition. (d) Pain *coming on without time relation to food* is characteristic of gastralgia.

*The effect of pressure* on the seat of pain helps us considerably in distinguishing structural from functional diseases of the stomach ; for whereas pressure relieves the pain due to the latter, the pain of organic diseases, such as cancer, simple ulcer, and gastritis, is aggravated by it.<sup>1</sup>

*Fallacies.* Pain of the acute type met with in gastralgia may be

<sup>1</sup> Two tender spots may be associated with stomach disease, one over the spine at the back, and one in front over the epigastrium.



mistaken for *biliary colic*, but there the pain is greater on the right side, and is sometimes followed by jaundice. In *hepatic* disorders, pain is more often limited to the right hypochondrium. The spine should always be examined for *caries*, especially when stomach pain is complained of by children. The pain in such cases is referred to the terminations of the intercostal nerves. The gastric crises of *tabes dorsalis* may be mistaken for simple gastritis. Pain in the *chest* (§ 24) must not be mistaken for stomach pain. *True angina pectoris* might be mistaken for that type of dyspepsia where the stomach is distended with gas and hampers the heart's action. Darting or lancinating pain may be due to *growths* involving the nerves near the stomach. In acute *pancreatitis* there is extreme pain of sudden onset in the left hypochondrium, and the case usually terminates fatally in a few days. Other pancreatic diseases are also attended by pain.

§ 192. **Nausea** or **vomiting** is, after pain, the most frequent and most definite symptom of stomach disorders, though it arises, also, in many other conditions. Its causes may be grouped under three headings :--(a) Local Causes, (b) Reflex Causes, and (c) Toxic Causes. Water-brash (*vide infra*) is sometimes spoken of by the laity as "vomiting," but this is not true vomiting. Regurgitation from a dilated œsophagus is another fallacy.<sup>1</sup> The mechanical discomfort of prolonged coughing may induce vomiting. Phthisical patients may come complaining only of the vomiting, and the physician may be led in consequence to treat the stomach instead of the lungs.

(a) LOCAL CAUSES, producing vomiting, may arise from (1) *Errors of diet*, such as shell-fish, tinned food, excess of alcoholic and other irritating foods. Under these circumstances the vomiting of the peccant material occurs with considerable rapidity. (2) *Irritant and corrosive poisons* and *emetics* also speedily give rise to vomiting. The diagnosis of this cause is aided by (i.) an examination of the vomit, which should *always be preserved*; it may smell of phosphorus (which is luminous in the dark), or of carbolic, or other acids.

<sup>1</sup> How closely regurgitation from the œsophagus, especially when it is dilated, may simulate vomit from the stomach is evidenced by 3 cases narrated by Dr. J. S. Bristowe (*Clin. Lects. and Essays on Dis. of the Nervous Syst.*, pp. 42 *et seq.*). The chief differential features are the ease and promptness with which food is returned from the œsophagus in cases of dilatation and spasm, the *absence of an acid reaction* in the matters so returned, and the absence of signs or symptoms definitely referable to the stomach.

(ii.) An examination of the mouth for any corrosive action. (iii.) The occurrence later of the toxic effects peculiar to the several poisons ; and (iv.) a history of poisoning obtained from the patient or his friends. (3) *Fermentation* of the contents of the stomach, such as that met with in dilatation, when the vomiting may occur at very considerable intervals, sometimes of a day or two ; the vomited matter also is frothy and contains *sarcinæ* and yeast (Fig. 63, p. 354). (4) *Diseases* such as acute gastritis, cancer, and simple ulcer, are usually accompanied by vomiting. In chronic gastritis the vomiting is of mucus and occurs in the early morning.

(b) REFLEX CAUSES may be classified under two groups,—(a) those due to cerebro-spinal irritation, and ( $\beta$ ) those due to visceral or sympathetic irritation.

(a) Reflex causes due to CEREBRO-SPINAL IRRITATION. 1. In *Hysterical vomiting*, the vomiting may follow any or every kind of food, no matter what its quantity or quality may be ; or perhaps digestible articles like milk will cause vomiting, while indigestible things like cheese may be retained. Sometimes this vomiting resembles a simple regurgitation, as compared with the urgent vomiting of organic disease, the symptoms of which are wanting.

2. In *Migraine*, and *Bilious headache*, the patient perhaps awakens with a headache and vomits only bile (merely an indication that the vomiting is urgent, or that the stomach is empty) ; the headache being relieved by the sickness (Chapter XIX.).

3. Another important cause of vomiting is *cerebral disease*, *e.g.*, tumour, early meningitis, abscess, Ménière's disease. This is recognised by : (i.) the vomiting occurs without relation to food ; (ii.) there is no nausea ; (iii.) the vomiting may be excited by simple change of posture ; (iv.) the presence of other cerebral symptoms, such as vertigo and perhaps optic neuritis (Chapter XIX.). Vomiting may also attend the gastric "crises" of *locomotor ataxy*. It occurs at intervals, and is usually severe. It is recognised by the absence of the knee-jerk and the presence of other symptoms of the disease.

( $\beta$ ) Reflex vomiting due to SYMPATHETIC OR VISCERAL IRRITATION may be met with in a great many abdominal disorders, such as peritonitis, pancreatitis, intestinal, biliary, or renal colic ; in all

stages of intestinal obstruction, and in strangulated hernia. It occurs also with pregnancy, uterine and ovarian disorders. If at the end of an operation the surgeon puts in stitches while the patient is coming out of the anæsthetic, vomiting is at once excited every time the needle is put in. This is especially noticeable with children.

(c) TOXIC CAUSES such as uræmia, Bright's disease, jaundice, and some of the acute specific fevers are accompanied by vomiting, especially at their advent. The vomiting of Addison's disease and pernicious anæmia might perhaps come under this heading.

The *treatment* of vomiting must of course be directed to its cause; but there are certain measures which can be applied to relieve the symptom. The patient should be kept absolutely at rest in the horizontal position, and without food, or only given milk in small quantities at a time, and iced water. Milk diluted with barley water, whey, or peptonised milk are given where ordinary milk is not retained. Among the remedies which may be employed are—effervescing mixtures, alkalies, hydrocyanic acid, bismuth, drop doses of vin. ipecac., opium, and acetanilid (especially in the vomiting after anæsthetics), seidlitz powder (if the vomiting be due to constipation), or calomel. Bromides and hydrocyanic acid are useful for nervous vomiting; a mustard leaf applied to the epigastrium may also be useful. For *Sea Sickness*, chlorobrom, bromides, chloral, morphia, and a number of patent remedies are recommended.

§ 193. *Hæmatemesis* (vomiting of blood). Bleeding from the stomach, if in any quantity, is usually accompanied by nausea and vomiting. And in the first place it is important to decide whether the blood really comes from the stomach.

*Sources of fallacy.* (1) Blood from the *lungs* may be mistaken for blood from the stomach (see *Hæmoptysis*, § 76). (2) *Epistaxis*, the blood running down the gullet and being vomited, is a common fallacy in children, in whom the blood is apt to be swallowed. It is recognised by making the patient blow his nose. In *epistaxis* there are no abdominal symptoms. (3) Blood from the *fauces* or *gums*, especially when the gums are spongy, or when *pyorrhœa alveolaris* exists, may give rise to a sanguineous vomiting or expectoration, the cause of which is very apt to be overlooked, if

unsuspected, even by competent observers<sup>1</sup> (§ 146); but the blood is mixed with saliva, and is rarely very large in amount. On the other hand, *hæmorrhage from the stomach* is (i.) preceded by a feeling of faintness and nausea; and (ii.) followed by melæna (tarry stools). (iii.) Blood from the stomach is mixed with food, and mostly brown; though it may be red if the quantity is large (*e.g.*, in ulcer), or if food has been brought up before the blood. (iv.) There is an absence of previous history or local signs of pulmonary disease, and there may be a previous history of disease or derangement of the stomach or liver.

The *causes of hæmatemesis* may be roughly divided into (a) those which produce a slight or protracted hæmorrhage; and (b) those giving rise to a large quantity at one time.

(a) **Slight or protracted hæmorrhages** occur chiefly in Chronic Gastritis, and Cancer. A temporary irritation or congestion of the stomach produced by irritating articles in the food, or by urgent vomiting, may be attended by *streaks* of blood in the vomit.

I. CHRONIC GASTRITIS, or gastric catarrh, is known by:—(i.) vomiting in the morning—often viscid mucus streaked with blood—or at other times. (ii.) It may be accompanied by, and due to disease of the liver (portal obstruction), or advanced cardiac disease, and is found especially in alcoholic subjects. Its other causes are mentioned in § 210.

II. CANCER OF THE STOMACH is recognised by:—(1) the patient is usually beyond middle age; (2) pain is complained of severe, constant, and generally worse after food; (3) the blood vomited is rarely copious, but typically “*coffee-ground*” in character, and may continue for weeks<sup>2</sup>; (4) the hæmatemesis is very rarely followed by melæna, because the blood is scanty, and because there is often obstruction of the pylorus; (5) progressive cachexia is very marked, and an abdominal tumour, or evidence of cancer elsewhere, may be found. (See also § 209.)

(b) A **large hæmorrhage** at one time may occur in Simple Ulcer of the Stomach or Duodenum; Liver Diseases; Other

<sup>1</sup> Cases of this kind, mistaken at first for organic disease of the stomach and the lungs, have been recorded. See Report Roy. Med. Chir. Soc., *Lancet*, June 16, 1900.

<sup>2</sup> This coffee-ground character is due to the digestion of the blood, which is constantly oozing into the stomach.



causes of Portal Obstruction; Aneurysm of the Aorta; Vicarious Menstruation; Morbid states of the Blood; or Chemical Irritants.

III. SIMPLE ULCER OF THE STOMACH. This is known by:— (1) the hæmatemesis is copious, therefore the blood is bright red, after first being a little black, and melæna usually follows; (2) characteristic pain occurs directly after food, and is *relieved* by vomiting; (3) it is found chiefly in young women, (4) who are the subjects of anemia, but not often great emaciation; (5) a history of previous attacks of bleeding is often present (§ 208).<sup>1</sup>

IIIa. ULCER OF THE DUODENUM is often difficult to distinguish from gastric ulcer. It occurs mostly in men.

IV. LIVER DISEASE (by causing portal obstruction) especially CIRRHOSIS (§ 252). The hæmatemesis may be slight, but it is more often very copious—the most copious met with.

V. OTHER CAUSES OF PORTAL OBSTRUCTION (see § 235), *e.g.*, tumour pressing on the portal vein. This, as with cirrhosis, is known by the other symptoms of such disease, *e.g.*, (1) the accompanying and rapidly increasing ascites, and (2) diarrhœa.

VI. ANEURYSM OF THE AORTA, or of one of its branches, leaking into the bowel, or œsophagus. This is known by (1) possibly a previous history of aneurysmal symptoms (§ 56); (2) the blood is copious; (3) sudden death is the usual result. This is the usual course, but in certain other cases there is a small recurrent leakage from the aneurysm, for a few days or weeks preceding death.

VII. VICARIOUS MENSTRUATION. It is impossible to be certain in the diagnosis of this condition. Its leading features are that it occurs periodically, and in women with amenorrhœa.

VIII. MORBID CONDITIONS OF THE BLOOD, such as yellow fever, malignant forms of the specific fevers, purpura, leukæmia, and hæmophilia.

IX. CHEMICAL IRRITANTS (*e.g.*, mineral acids), or mechanical injuries from articles which have been swallowed.

In the *differentiation* of the causes of hæmatemesis (1) examine the stomach; (2) examine the liver especially for cirrhosis, which is perhaps the commonest cause of hæmatemesis, simple or

<sup>1</sup> Cases have been recorded of profuse hæmatemesis resembling that of simple ulcer, occurring in older patients, which disappeared under antisyphilitic treatment. The condition was apparently a syphilitic ulcer of the stomach. Dalgleish, *Lancet*, 1898, ii., p. 110.

malignant ulcer being the next ; (3) ascertain the approximate quantity of vomited blood, and then review the case.

*Prognosis.* Hæmatemesis is usually a serious symptom, but its gravity depends upon the cause. In portal congestion, hæmatemesis not infrequently serves as a safety-valve to relieve the abdominal congestion, and in a sense is beneficial. As regards the lesion, aneurysm is the most grave of the causal conditions ; then, in order, cancer, morbid blood states, cirrhosis, and simple ulcer. The amount of hæmorrhage is a less valuable guide to prognosis ; although where the amount is copious the patient will remain debilitated for a considerable time.

The *treatment* of hæmatemesis must also have reference to the cause. (a) When small in quantity it calls for but little immediate treatment. (b) When in larger amount, the patient must not be moved from the place where the bleeding occurred, and should be kept absolutely at rest in the horizontal position. Nothing should be given by mouth except iced water for some time ; feed by rectum. Opium is given for the pain. If bleeding continues give astringents, such as alum (gr. v.) and acid. sulph. dil. (℥ xx.), or ergot. In profuse hæmorrhages saline transfusion may be necessary. If the bleeding is due to vicarious menstruation, try to restore the natural discharge by giving iron and aloes, and by hot sitz- and foot-baths. Supra-renal gland has recently been advocated as a remedy.<sup>1</sup>

§ 194. The **other local symptoms** of gastric disorder are of considerable diagnostic value.

1. **BAD TASTE IN THE MOUTH** is very often complained of in gastric disorders, and is always most noticeable in the morning. **DRYNESS OF THE LIPS** is another very constant manifestation, and will often give an acute observer the first clue to the existence of gastric disorder.

2. **THIRST** is often associated with dyspepsia ; it is specially apt to occur with dilatation of the stomach, inflammatory stomach lesions, and in all cases where there is persistent vomiting.

3. **FLATULENCE** is a distension of the stomach or intestines by gas, which may be brought up by the mouth or passed by rectum. This gas may be due to repeated swallowing of saliva and air, as

<sup>1</sup> *Brit. Med. Journ.*, Nov. 3, 1900.

in chronic gastritis<sup>1</sup>; or to decomposition of food. Among its causes are excessive ingestion of vegetables, sugars, and starches, chronic dyspepsia, or chronic gastritis, and all conditions attended with dilatation of the stomach.

4. "HEARTBURN" and ACID ERUCTATIONS are usually met with together. Heartburn is a burning sensation passing up from the epigastrium to the pharynx; and sometimes mouthfuls of acid fluid are brought up at the same time. It is due to hyper-acidity and partial regurgitation of the gastric contents into the lower end of the œsophagus.

*Causes.* Hyper-acidity, or "acid risings," may be of two kinds. (a) *Organic acids* are met with in diseases where there is *deficient* gastric secretion—some forms of atonic dyspepsia, chronic gastritis, cancer, and dilatation of the stomach. HCl is a germicide, and when from any cause it is absent, bacteria flourish; fermentation ensues within a few hours after food, and is accompanied by pain in the epigastrium. The three principal types of acid fermentation are: butyric, lactic, and acetic.

(b) Hyperchlorhydria or *excessive secretion of HCl*. This condition is met with in one form of acid dyspepsia, and chronic glandular gastritis. The name is often misapplied to the acidity due to organic acids arising from fermentation. Here, the pain or "gnawing" generally occurs *before* meals, and is temporarily relieved by food. (See also § 206.)

5. "WATER-BRASH" (pyrosis) is the name given to a clear alkaline fluid expelled from the mouth in gushes, most often in the morning. Sometimes it is expelled without any kind of straining, but more often it is attended by retching. It is probably a reflex hyper-secretion of saliva due to irritation in the stomach, swallowed during the night. It is met with in many dyspeptic conditions, and is a fairly constant symptom in *chronic gastritis*.

6. ANOREXIA (loss of appetite) is not always an indication of stomach disease, as it is present in many general constitutional disturbances. Its chief clinical importance lies in its presence in the earliest stage of *gastric cancer*. In *atonic dyspepsia* there is

<sup>1</sup> The semi-voluntary swallowing or gulping down of air is met with in lunatics, and in some hysterical or neurotic individuals without gastric derangement. It is diagnosed from dyspepsia by the absence of all other symptoms of that condition.

sometimes no appetite before a meal ; but the first few mouthfuls of food induce secretion of gastric juice, and so excite appetite. HYSTERICAL ANOREXIA is known by—(i.) The appetite is perverted ; for instance, the patient will only eat some unreasonable article, *e.g.*, a penny bun bought at a particular shop.<sup>1</sup> Such patients may push matters to extremes, almost to the point of death. The condition is really a form of hysterical insanity. (ii.) It is only met with in the female sex, in whom also (iii.) the hysterical stigmata are generally present (Chapter XIX.).

INCREASED APPETITE is far more often met with, as Shakespere pointed out, in gastric disorders. It is found in some cases of chronic dyspepsia, chronic gastritis, and dilated stomach, in pregnancy, and during convalescence. A FALSE APPETITE which is satisfied with the first few mouthfuls of food is sometimes met with in sub-acute and chronic gastritis, owing to the irritated condition of the mucous membrane. BOULIMIA or ravenous appetite is seen in diabetes, in neuroses of the stomach, after acute gastritis, in wasting disorders such as mesenteric gland disease, in phthisis, intestinal worms, and Graves' disease.

§ 195. **General or remote symptoms** are very constant accompaniments of all gastric diseases.

1. GENERAL MALAISE and a sense of ill-health and incapacity for work are among the earliest and most constant accompaniments of all derangements of the digestion, whether functional or organic. The dark rim beneath the eyes, and the sallow, "earthy" complexion, so frequently associated with town-dwellers, is quite as often due to dyspepsia, just as this latter is often due to defective teeth or to the insufficient use of them. EMACIATION is not so frequently associated with gastric disorder as might be supposed, though in very chronic cases there is sure to be some loss of flesh. Early and marked emaciation is, however, one of the surest indications of cancer of stomach.

2. The CARDIAC SYMPTOMS met with in dyspepsia are—Palpitation ; pain in the region of the heart (pseudo-angina) ; dyspnoea and asthmatic paroxysms<sup>2</sup> ; syncope and vertigo ; intermission of

<sup>1</sup> An instance of this kind has happened in my experience, and the patient, thin as a skeleton, was really on the point of death when I first saw her.

<sup>2</sup> True asthma is sometimes occasioned by an attack of indigestion.



the cardiac rhythm; and cough, due to pharyngeal catarrh or reflex irritation. Collectively, these symptoms may, as previously mentioned, give rise to the impression that the case is one of C. V. D., although the heart may be structurally healthy.

3. FUNCTIONAL DISTURBANCE OF NERVOUS SYSTEM:—*Headache and depression of spirits* are invariably met with in all forms of dyspepsia. A sense of general ill-health and irritability of temper out of all proportion to the local mischief attends most gastric disorders, and, where stomach symptoms are not prominent, may lead the physician away from the true cause.<sup>1</sup> Any or all of the symptoms of *neurasthenia* (Chapter XIX.) may undoubtedly result from gastric disorder; and this constitutes one variety of what the author has described as Toxic Neurasthenia.<sup>2</sup>

4. DIARRHŒA may accompany stomach disease when the gastric contents are of an irritating nature; CONSTIPATION is usually found with simple ulcer, cancer, and chronic gastritis. But a more usual condition is an IRREGULARITY of the bowels, accompanied by borborygmi (rumbling in the bowels).

5. The URINE invariably exhibits signs which reveal the disturbances in the metabolism of the body. The commonest of these, perhaps, is an excess of URATES, as shown by the pinkish sediment when the urine cools. In other cases PHOSPHATES form the deposit; and in certain cases OXALATES are found (compare § 315). In these circumstances dyspepsia must be regarded as a predisposing cause of renal and vesical calculus.

6. SKIN SYMPTOMS:—General *pruritus* may accompany many forms of gastric derangement. *Flushing* of the face after meals is met with in many gastric disorders, especially when they occur in the female sex. The face may be swollen so that the case appears like one of acute Bright's disease; but the sudden onset, without much constitutional disturbance, and early disappearance

<sup>1</sup> Gautier and others have shown that certain toxins are being constantly generated within the body; and especially in the digestion and metabolism of the food. They produce no evil effect in health when not in excess, partly because they are excreted by the urine, feces, and sweat, partly because they are being constantly destroyed (chiefly perhaps by the liver). But when in excess they produce profound disturbance of the general economy, and especially of the nervous system. Their composition as far as we know is analogous to the vegetable alkaloids (morphine, atropine, etc.), and they are therefore known as *animal alkaloids*. There are 2 kinds—(1) *Plomaines*, which are produced by the fermentative disintegration of dead albuminous substances (e.g., during digestion); and (2) *Leucomaines*, which are produced by the activity of living nitrogenous substances. (See also Lander Brunton in the *Practitioner* for Oct. and Nov., 1880.)

<sup>2</sup> "Clinical Lectures on Neurasthenia," Glaisher, London.

on curing the indigestion, distinguish it from that disease. The occurrence of general *urticaria* in certain individuals after eating indigestible articles is very common. It may also attend the different forms of gastric disorder.

#### PART B. PHYSICAL EXAMINATION.

Disorders of the stomach are investigated by Inspection, Palpation, Percussion, and Examination of matters vomited, or withdrawn from the stomach by a tube.

§ 196. **Inspection.** (1) The *Teeth* in all cases must be closely examined. Among my out-patients the two commonest causes of indigestion are certainly defective teeth, and bolting the food. Disorders of the teeth are referred to in § 144.

(2) The *Tongue* and its diseases have been already described, and § 148 should be specially consulted. At one time the tongue was thought to indicate the state of the stomach, but this is by no means always the case, and it is a far more certain indication of the patient's general condition. But even in this, allowance has to be made for certain variations, namely: (i.) individual variations, since a coated tongue is normal to some even in health, and a clean tongue in others may be associated with disease; (ii.) certain diets, *e.g.*, milk, produce a coated tongue; and (iii.) certain habits, *e.g.*, smoking and "tippling," also coat the tongue.

(3) Inspection of the epigastric region may reveal a tumour, or the peristaltic movements of a dilated stomach.

§ 197. **Palpation.** To palpate the stomach successfully requires considerable experience. The patient's shoulders should be supported, and he should be instructed to open his mouth, to draw up his knees, and to "let his breath go."<sup>1</sup> Talking to him is useful to distract his attention. The hand should be warmed and it should always be laid quite *flat* upon the abdominal wall. Then only can we detect the presence of a tumour, tenderness, or other abnormality.

*Gastric succussion* or *splashing* is made out by placing one hand on each side of the stomach, and suddenly pressing inwards the

<sup>1</sup> Some say it is better to have the legs extended loosely; and some advise examination in a hot bath to relax the muscles. Finally anaesthesia with chloroform or ether may be necessary in very obscure cases.

finger tips of each hand alternately. Listening over the stomach with a binaural stethoscope during this procedure materially aids in discovering this sign. Splashing can be *normally* elicited during the process of digestion, *i.e.*, during the first hour or two after a meal, especially if much fluid has been taken. But, if succussion can be elicited after that time it suggests that there is atony of the stomach, either with or without dilatation.

§ 198. **Percussion** of the stomach is not very satisfactory or precise. The only diseases in which the area of stomach resonance has to be defined are DILATATION (§ 211) and GASTROPTOSIS (§ 213).<sup>1</sup>

**PERCUSSION OF THE STOMACH.** The normal stomach is depicted in Fig. 74, and its situation in Fig. 62, the cardiac orifice being under the 7th costal cartilage, about an inch to the left of the sternum. The pylorus is just to the right of the sternum, and 2 inches below it. The lesser curvature corresponds closely to a line drawn round the tip of the xiphoid cartilage and along its left side. The fundus of the stomach is its highest point, and is just behind the heart apex at the fifth rib. The position of the greater curvature (lower border) of the stomach varies according to the degree of distension; it ought not to come lower than midway between the umbilicus and xiphoid. Its position is hard to define, owing to the proximity of the transverse colon. Only the lower border and part of the anterior wall of the stomach are normally in contact with the abdominal parietes. The percussion note over the stomach is tympanitic, but has rather a lower pitched tone than that over the transverse colon, which is of course also resonant.

The rough outline of the stomach resonance can be made out by percussion in the usual way without any elaborate precautions, but the following method is more accurate. The stomach being empty, let the abdomen be stripped, with the patient standing. Percuss lightly from above downwards and mark the change of note, the stomach being usually the most resonant of the abdominal viscera. After this, give the patient a large drink of water. The note over the lower border of the stomach is now dull; and the lower level of the dull note so produced can be marked. Finally, the area of the stomach can be percussed out, with the patient in the recumbent posture, when the lower boundary will be found at a different level.

*Ausculto-percussion* is sometimes employed to define more accurately the boundaries of the stomach. Place the stethoscope in the angle between the xiphoid cartilage and the l. costal margin, and elicit the normal stomach note by percussing lightly around it. Then percuss up from the pubes towards the stethoscope till a note is heard similar to that first elicited. When there is fluid in the stomach, the percussion note varies with the position of the fluid, and it is then necessary to percuss the boundaries, first with the patient lying on his back, then on his right, and finally on his left side.

*Fallacies.* (1) Apparent enlargement of the stomach may arise in

<sup>1</sup> GASTROPTOSIS, *i.e.*, a dropping or dislocation of the stomach, undoubtedly occurs occasionally, but it requires considerable skill and experience to make the diagnosis.

contracted cirrhotic liver, or fibrosis of the lung. (2) Apparent *diminution* may occur when the liver is enlarged or pleuritic effusion is present. (3) *Dislocation* of the stomach downwards may simulate dilatation. In such cases the lesser curvature may sometimes be seen or felt below its normal position.

§ 199. **Motor Insufficiency of the Stomach** (Gastric Atony or Myasthenia) leads to dilatation (§ 211). The methods of detecting motor weakness of the stomach form the most important additions to our knowledge in recent years. A man can live and maintain weight without the secretory and resorptive functions of the stomach being quite perfect; but serious auto-toxic effects and malnutrition result from a *retention of food* within the stomach. Moreover, motor insufficiency is always attended sooner or later by disturbance of the secretory and resorptive powers.

1. LEUBE'S METHOD of estimating the duration of gastric digestion consists of the administration of a normal meal of 50 gm. bread, 200 gm. beefsteak, and a glass of water. Six to seven hours is the average time required to completely digest such a meal, and no solid portions should then be found in the stomach by the use of the tube (§ 200). There are, however, many physiological and individual variations.

2. EWALD'S AND SIEVERS' METHOD consists of the administration, with a normal meal such as the above, of 1 gramme of salol. This substance is not acted on by the acid contents of the stomach, but it is immediately decomposed by the alkaline juices of the duodenum into salicylic acid and phenol, and leads to the immediate appearance in the urine of salicyluric acid. The latter is recognised by the violet colour produced by the addition of ferric perchloride. Normally it will appear in the urine from 40 to 75 minutes after taking the salol (Hemmeter): delay indicates retardation of the passage of the stomach contents into the intestines. Various observers give as the limits, in health, 40 minutes to 2 hours. For this test the patient must pass water every 5 or 10 minutes; and the excretion of salicyluric acid depends on blood pressure, kidney function, and other conditions. Huber improved on this method by ascertaining when it *ceased* being excreted in the urine. Normally it ceases in 24 hours, but if gastric peristalsis be impaired it may continue to 48 hours or more. Others have used potassium iodide or sodium salicylate. Fleischer administered iodoform in a gelatin capsule during a meal and tested repeatedly for the appearance of potassium iodide in the saliva. All of these tests depend upon the same principle.

3. Klemperer introduced 100 gm. olive oil (which is not absorbed by the stomach) into the stomach previously washed clean by lavage. Two hours later the oil which remained in the stomach was removed and weighed.

4. Einhorn used an instrument which records the gastric movements by dots on a narrow piece of paper (*New York Med. Journ.*, Sept. 15, 1894).

5. Hemmeter used an intragastric bag internally, and a pneumograph externally, both connected with a recording kymograph (*Diseases of the Stomach*, p. 77).



§ 200. **Examination of Stomach Contents.**<sup>1</sup> First as to the CHEMISTRY OF DIGESTION, from a clinical standpoint, and the practical information to be derived from chemical examination of the stomach contents. *Four processes* normally take place in the stomach: (1) the conversion of starch into sugar, begun in the mouth, is carried on a stage further; (2) proteids are changed into peptones; (3) fat globules are set free from their envelopes; (4) milk is curdled. Delay in digestion may be caused by (1) deficient peristalsis of the stomach walls, (2) deficient quality or quantity of the gastric juice, (3) the consumption of indigestible articles, or (4) the dilution of the gastric juice by drinking too much fluid at meal-time.

The gastric juice contains HCl, water, pepsin, rennet, mineral salts and a little mucus. Pepsin and rennet exist in the secretory cells only as zymogens, which, in the presence of the HCl, become active ferments or enzymes. In the healthy state, as the result of digestion, about 30 c.c. of fluid should be obtained from the stomach one hour after Ewald's test-meal (*vide infra*), straw-coloured, without much odour, without organic acid, and with about 0.2 per cent. of free HCl.

As regards *hydrochloric acid*, much depends on the time of examination. Thus its presence in the stomach before breakfast, when there should be none, indicates hypersecretion. *Hyperchlorhydria* is merely a convenient term for excessive secretion of HCl. It has come to be somewhat loosely used for "excessive acidity"; and thus to be confused with the acidity of fermentation (due to organic acids). On the other hand, after a meal, a negative result on testing for HCl would indicate the absence of peptic activity, as an acid is required to convert the inactive proenzyme or pepsinogen into pepsin. An excess of HCl is distinctive of gastric ulcer, as compared with gastralgia; for in the latter, the HCl is normal or diminished. HCl is also diminished in all catarrhal conditions of the mucous membrane, in great anæmia and neurasthenia. When there is a difficulty in diagnosing malignant disease, the absence of free HCl is a point in favour of cancer.

*Lactic acid* is not normally present in the gastric juice after digestion has proceeded for one hour, but traces may be found due to the ingestion of lactic acid in certain foods, or to fermentation in the mouth. A decided reaction with Ueffelmann's test (*infra*) is found with cancer of the stomach—but a negative reaction does not have equal value in proving the absence of the disease.

*Butyric* and *acetic* acids prove the presence of fermentation, and are found where HCl is deficient, or the food is delayed in the stomach, as in dilatation of the stomach, or a narrowing of the pylorus.

The secretion of *pepsin*, according to most authors, is not interfered with, unless there be destruction of the glandular elements of the stomach. The presence of pepsin is also indicative of the presence of HCl (which is necessary to convert pepsinogen into pepsin), and of the activity of the glands. If only pepsinogen is found, the glands are active, but HCl is deficient. The presence of the latter (pepsinogen) is an important feature in diagnosing between chronic gastritis, where the glands are destroyed,

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It is not possible here to give more than a brief outline of this important subject, and the excellent treatises of Hemmeter and others on Diseases of the Stomach may be consulted with advantage.

and dyspepsia, or any neurosis of the stomach, where the glands are not destroyed.

*Pepsin* is diminished or absent in the later stages of gastritis and cancer. Its presence is a strong proof that there is no serious destruction of the glands of the stomach.

**SUMMARY.** The two most important questions, therefore, to determine in an analysis of stomach contents are: (1) The presence and amount of HCl; and (2) The presence and amount of pepsin and pepsinogen. The latter question is specially important because

- (a) If *pepsin is present*, the gastric glands are active and HCl is present, since this is required to convert pepsinogen into pepsin.
- (b) If *pepsinogen is present*, the gastric glands are active, but the HCl is deficient.
- (c) If *neither are present*, the gastric glands are destroyed or inactive.

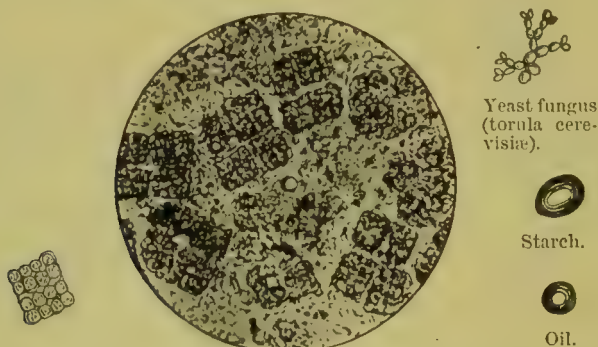


Fig. 63.—VOMITED MATTER. Sarcinæ Ventriculi,  $\times 350$ . These are fungoid vegetations which indicate that food residues are retained in the stomach and fermentation has taken place. Yeast fungus has a similar significance.

**Examination of Gastric Contents after a test-meal** is a useful method of investigation in cases of severe and intractable indigestion or dilated stomach. To the patient it entails considerable discomfort, and to the physician some trouble. It consists of three steps: (a) The administration of the test-meal; (b) The withdrawing of the gastric contents by means of a stomach tube; and (c) The microscopical and chemical examination of the material withdrawn.

(a) **The test-meals** suggested are of many kinds, but there are two which will answer all purposes—

1. *Test-meal of Ewald and Boas*.—Wheat bread 30–70 grms.; water 300–400 c.c. Give in the morning when the stomach is empty. Examine the stomach contents an hour later.

2. *Double test-meal of Salzer*.—Lean cold roast meat 30 grms. cut small in order not to obstruct the stomach tube. Milk 250 c.c., rice 60 grms., and one soft boiled egg. Four hours later, stale wheat bread 35–70 grms., and 300–400 c.c. water. Examine contents one hour later, and normally, there should be no remnants of the first meal found.

(b) **Method of passing the tube.** Use a rubber tube nearly 2 yards long, with large “eyes” and a funnel fixed to the end of it. It should be thoroughly cleaned, and moistened with warm water before being passed into the pharynx. The patient should sit with the head erect, the chin being projected forward and the mouth open. The tube is passed into the pharynx while the patient is instructed to swallow and the tube is pushed down into the stomach. Then bring the funnel end down to a lower level than the stomach, and the contents should syphon out into a receptacle. If the gastric contents do not flow at once the patient should strain as if trying to vomit; or draw out the tube a little lest the end becomes folded upon itself, or the “eye” stopped by a fold of mucous membrane.

It may be necessary to pour a small quantity of water into the funnel, and the moment the tube is filled with water nip it below the funnel and bring that end down to the receptacle. It is convenient to tie a thread round the tube in order to mark a position sixteen inches from its tip. By this means we know the amount of tube which has been passed into the œsophagus, and consequently when the tip ought to have reached the stomach. A flexible tube curling up in the œsophagus is a contingency which may thus be obviated.

Its use is *contraindicated* in angina, heart failure, fevers and other acute diseases, recent hæmorrhages, great arterio-sclerosis, aneurysms, and a high degree of emphysema and bronchitis.

(y) **Examination of Stomach Contents** after the test-meal.

**MICROSCOPICALLY** (Fig. 63) we can detect fat globules, starch cells, vegetable and muscle fibres, and sometimes fatty crystals, leucin and tyrosin, cells of the mucous membrane, torulæ cerevisiæ or sarcinæ.

**CHEMICALLY** we have to answer six questions: (a) Are the contents acid? (b) What is the total acidity? (c) What is the acidity due to? (d) Is pepsin present? (e) Is pepsinogen present? (f) Is rennet present?

(a) *Are the contents acid?* can be roughly detected by the use of carefully neutralised litmus paper.

(b) *Method of estimating Total Acidity*—i.e., acidity due to hydrochloric acid, organic acids, carbon dioxide, and such acid salts as react acid to phenol-phthalein. Titrate 10 c.c. unfiltered gastric contents with deci-normal solution of caustic soda (prepared free from carbon dioxide), using a 1 per cent. solution of phenol-phthalein as indicator. Add a drop or two of the indicator to the gastric contents; then run in the deci-normal caustic soda solution from a burette till further addition produces no alteration in colour. Normally 5 to 6 c.c. of deci-normal caustic soda are required for 10 c.c. of unfiltered gastric contents. For clinical purposes the total acidity is expressed in terms of an arbitrary scale by multiplying the number of c.c. of deci-normal soda required, by 10. Consequently the normal total acidity is 50 to 60. The acidity may be calculated *in terms of HCl* by multiplying the number of c.c. of the soda solution used, by 0·00364. The normal percentage of HCl actually present is, therefore,  $5\cdot5 \times 0\cdot00364 = 0\cdot2$  per cent.<sup>1</sup>

(c) *Methods of finding what the Acidity is due to.* The acidity of the gastric juice is normally due to free HCl, but in the gastric contents other acids are sometimes found. In abnormal conditions fatty acids are present, and organic acids, which are products of fermentation. HCl acts as an anti-fermentative agent, and the presence of organic acids after the first hour of digestion therefore indicates a deficiency of HCl.

(i.) *Are there any free strong acids?* Dip a piece of Congo-red paper into the filtered gastric contents, and the presence of free HCl or strong organic acids (as butyric, acetic, or lactic) will cause it to turn blue. Hydrochloric acid in combination with albumen does not cause this coloration, although it reacts acid to phenol-phthalein. The next question is to decide whether the free acid is mineral or organic, i.e., is it HCl on the one hand, or lactic, butyric, or other organic acid on the other hand?

(ii.) *Test for HCl.* Add a few drops of phloro-glucin-vanillin solution to an equal quantity of the filtered gastric contents in a porcelain capsule. Heat over a spirit lamp. A rose-red ring at the circumference of the fluid indicates presence of free HCl.

(iii.) *Tests for organic acids*—*Lactic acid.* To insure that no lactic acid is introduced with the food, it is usual to give Boas' meal, which consists of a plateful of oatmeal soup made by boiling down to one pint a quart of water with one tablespoonful of rolled oats. If the patient digests slowly give the soup, after thoroughly washing out the stomach, in the evening, and examine the contents in the morning. *Ueffelmann's test* for lactic acid.—Owing to the possible obscuring of the colour by other acids, it is best to dissolve out the organic acids by shaking up 10 c.c. filtered gastric juice with 100 c.c. of neutral sulphuric ether for a few minutes. Divide into two portions. Evaporate one portion over a water-bath and make the residue up to 20 c.c. with distilled water. Then add 10 c.c. of a 4 per cent. sol. carbolic acid, and 1 drop of liq. ferri perchlor.: an amethyst-blue colour is obtained in the absence of lactic acid. A lemon or canary-yellow hue appears if lactic acid be present, even in so small a quantity as 0·1 per cent. The second portion should be allowed to evaporate at room temperature because of the volatile properties of butyric and acetic acids, for which we now proceed to test. *If Butyric acid be present*, Ueffelmann's test gives a turbid brown precipitate. On adding a trace of  $\text{CaCl}_2$  to the extract, oily drops separate out, with the characteristic rancid odour. *Acetic acid.*—Neutralise the ethereal extract with caustic soda, and add a little of a very dilute solution of liq. ferri perchlor. If acetic acid be present a claret-red colour is produced, and boiling causes a cloudiness or even a precipitate if the quantity of acid be not too small.

(iv.) *Method of estimating total amount of HCl, free and combined with albumen, in the gastric juice* (Töpfer). The absence of free HCl does not prove there is no secretion of HCl,

<sup>1</sup> In a recent publication (Münch. med. Wochschr. 1900, pp. 381–382) Cohnheim and Krieger direct the estimation of total acidity with phenol-phthalein, as above, followed by estimation, also with phenol-phthalein, of total acid remaining in 10 c.c., from which combined HCl has been precipitated by the addition of calcium phosphotungstate. The difference gives combined HCl.

for it may be present combined with the albumen in the gastric contents; and it is useful sometimes to ascertain this. Take three separate portions of filtered gastric contents, each 10 c.c. In the first ( $\alpha$ ) find the *total acidity* (*vide supra*), using 1 per cent. solution phenolphthalein as an indicator. In the second ( $\beta$ ) find the *acidity due to free acids* by using alizarin as an indicator, and titrating with deci-normal soda solution, till a pure violet hue is evolved (this violet hue must be the shade obtained by adding 3 drops of alizarin to 5 c.c. of 1 per cent. solution of sod. carb.). This reagent does not react with the loosely combined HCl, and therefore the difference between  $\alpha$  and  $\beta$  gives the amount of loosely combined HCl. ( $\gamma$ ) In the third portion test for free HCl, estimating it with 0.5 per cent. alcoholic solution dimethyl-amido-azo-benzol, or preferably methyl orange, as an indicator. Titrate with deci-normal solution till the red colour is lost which denotes the presence of HCl, and the yellow appears which denotes the absence of free HCl. This gives the amount of *free HCl present*. The difference between  $\beta$  and  $\gamma$  gives the acidity due to organic acids, whilst the difference between  $\alpha$  and  $\beta$  added to  $\gamma$  (*i.e.*,  $\alpha - \beta + \gamma$ ) gives the total hydrochloric acid, both free and in combination with albumen.

(d) *Is pepsin or pepsinogen present?* The cells of the gastric glands secrete pepsinogen, and, in the stomach, HCl converts this into pepsin. A very small quantity of pepsin is needed to digest large quantities of albumen. Test for *pepsin* by setting aside 25 c.c. of filtered gastric juice (if HCl has previously been found present in it) and keep at a temperature of  $37^{\circ}$  to  $40^{\circ}$  C., with the addition to it of fibrin or coagulated egg-albumin cut very small. If HCl be absent, a few drops must be added. 0.05 grm. should be digested in 3 hours.

(e) *Is pepsinogen present?* Pepsinogen cannot exist in the presence of free HCl as it at once becomes converted into pepsin. If, therefore, the gastric contents are found to contain no HCl, pepsinogen may be detected by adding 3 or 4 drops of dilute HCl, and then proceeding to test for pepsin as directed in (d), *ante*.

(f) *Is rennet present?* Rennet or Chymosin can act in a neutral or a feebly alkaline medium. It is the milk curdling ferment. (i.) To test for the *Enzyme*, treat 5 to 10 c.c. of sterile milk with 5 drops of filtered gastric juice, and keep at a temperature of  $38^{\circ}$  C. for a quarter of an hour. If coagulated, then rennet enzyme is present. (ii.) Test for the *Zymogen*. In the absence of HCl, a lime salt will act equally well to convert the zymogen in the rennet into an active enzyme. Thus, by treating an alkaline or neutral juice (rendered alkaline or neutral if necessary) with 2 c.c. of a 1 per cent. solution  $\text{CaCl}_2$  at a temperature of  $38^{\circ}$  C. the milk will be coagulated in a few minutes if the zymogen be present.

(g) Mucus in the vomited matter or gastric contents may be detected generally by the slowness with which it filters. Only when in quantity does it become visible, but its detection is greatly aided by mixing a suspected portion with water, when the mucus can be seen floating like jelly. The presence of mucus in any quantity is pathognomonic of gastritis.

### PART C. DISEASES OF THE STOMACH, THEIR DIFFERENTIATION, PROGNOSIS, AND TREATMENT.

§ 201. The routine investigation of the disorders of the stomach consists of 3 steps:—

FIRST: We must identify the patient's LEADING SYMPTOMS as being referable to gastric disorder (see Part A.).

SECONDLY: Inquire as to the HISTORY, and especially whether the illness came on *acutely* and recently, or whether, as is more usual, it came on insidiously, and has run a *chronic* course. Much depends on the skill and method with which the history is elicited. Inquire particularly as to pain or discomfort and its relation to meals, and as to the other symptoms mentioned in Part A.

THIRDLY: Proceed to the PHYSICAL EXAMINATION, and ascertain whether there be any localized tenderness and pain; and whether any tumour or other abnormality be present.



## Classification of disorders of the stomach.

### A. Acute Diseases of the Stomach.

I. Acute Dyspepsia (Bilious Attack) : *without tenderness* . . . § 202

II. Acute Gastritis : *with tenderness* . . . . . § 203

### B. Chronic Diseases of the Stomach.

a. WITHOUT TENDERNESS ON PRESSURE : and pain less marked.

I. Chronic Atonic Dyspepsia . . . . . § 205

II. Chronic Acid or Irritable Dyspepsia . . . . . § 206

III. Gastralgia (Gastric Neuralgia). . . . . § 207

b. WITH TENDERNESS ON PRESSURE : pain a marked feature.

IV. Simple Ulcer of the Stomach . . . . . § 208

V. Cancer of the Stomach . . . . . § 209

VI. Chronic Gastritis . . . . . § 210

c. DILATATION OF THE STOMACH . . . . . § 211

This classification, based on the presence or absence of tenderness, is not very satisfactory, and each group will be found to contain many exceptions. It is, however, the least unsatisfactory of those classifications possible at the present time. The division of Chronic Dyspepsia into Atonic, and Acid, is also very unsatisfactory. See also footnote, p. 360.

If the patient's symptoms have come on gradually, and lasted a considerable time, turn to **Chronic Disorders** of the Stomach, § 204.

If, on the other hand, his symptoms have commenced somewhat suddenly and recently, the case is probably one of the two **Acute Disorders** of the Stomach : I. ACUTE DYSPESIA ; or, II. ACUTE GASTRITIS.

I. *The patient—whose temperature is normal—complains of NAUSEA, GASTRIC DISCOMFORT, headache, and depression, which have come on suddenly ; and there is no marked epigastric tenderness—the disease is probably ACUTE DYSPESIA.*

§ 202. **Acute Dyspepsia** (" bilious attack ") consists of a sudden disturbance of the digestion in a previously healthy person, such as occurs in association with surfeit, high living, or other errors in diet.

The *symptoms*, which come on suddenly, are—(1) Pain, or a feeling of oppression or distension, in the epigastrium, occasionally accompanied by some tenderness on pressure, though the tenderness is never very marked. (2) Nausea and vomiting very generally ensue (but not always). (3) Headache, depression, anorexia, coated tongue, constipation, scanty urine loaded with lithates.

(4) The illness is sometimes preceded and accompanied by drowsiness, and not infrequently there is a history of previous similar attacks.

The *diagnosis* is not difficult, the only condition resembling it being acute gastritis, in which the constitutional symptoms are more apparent, the duration of the illness considerably longer, and the *tenderness much more marked*. Irritant poisoning comes on much more suddenly with very urgent vomiting (§ 192).

*Etiology.* (1) Too large a meal, especially after previous fatigue. (2) Errors in diet, such as excess of alcohol (which retards digestion), ice, and many other articles which vary with the idiosyncrasy of the individual.

*Prognosis and Treatment.* Acute dyspepsia of the kind here referred to usually passes off in two or three days. (1) If pain be present, assist vomiting by mild emetics, such as copious draughts of salt and water, tickling the fauces, etc. Violent emetics aggravate the condition. (2) Three grs. of calomel, and milk diet for a day or two, generally relieve the condition. (3) Bismuth and tonics may be given during convalescence.

II. *The patient complains of considerable PAIN or discomfort, and TENDERNESS IN THE EPIGASTRIUM, with nausea or vomiting, all of which have come on somewhat suddenly*—the disease is probably ACUTE GASTRITIS.

§ 203. **Acute or sub-acute gastritis** is relatively a much more serious disorder than the foregoing. It consists of a sudden derangement of digestion due to inflammation of the stomach. This condition is not so much a catarrhal inflammation of the mucous membrane (excepting in cases of irritant poisoning) as of the glands of the stomach.

*Symptoms.* (1) Pain, intense and burning, or a feeling of distension in the epigastrium, coming on directly after food, and accompanied by tenderness on pressure. (2) Vomiting, not always immediately after a meal, of undigested food, sometimes with streaks of blood. (3) Malaise, anorexia, slight pyrexia, headache, depression, and other constitutional symptoms may be present, attended sometimes by great prostration, thirst, furred and coated tongue. (4) Diarrhœa may ensue after a day or two.

The *diagnosis* may have to be made from acute dyspepsia (§ 202), and from the other causes of Vomiting (§ 192).

Recovery generally takes place in about 3 to 6 days, the affection rarely lasting longer than 8 or 10 days. It may go on to chronic gastritis. Death rarely takes place, excepting from irritant poisoning, or in cases of membranous gastritis.<sup>1</sup>

*Etiology.* (1) In the majority of cases simple acute gastritis is caused by errors in diet, or by decomposing meat, *e.g.*, tinned food containing ptomaines<sup>2</sup>; an excessive quantity of normal food will cause it. (2) Irritant poisons (*e.g.*, arsenic, antimony, phosphorus, etc.). In long continued vomiting, without apparent cause, poisoning should be suspected, and the vomited matters examined. (3) In some cases, gout and other constitutional conditions predispose to or determine an attack.<sup>3</sup> Heart, lung, and liver disease are predisposing causes.

*Treatment.* The indications are:—(1) To remove any irritant that may be present from the stomach. This can be done by promoting vomiting, which is specially indicated if the epigastric pain continues, employing the means mentioned in § 202. It may be desirable to give a purgative, such as 3 grs. of calomel, and a seidlitz powder in the morning. Hot fomentations or a mustard leaf to the epigastrium may relieve the pain. (2) The second indication is rest to the stomach, which is gained by 12 or 24 hours' abstinence from food, followed by milk in small quantities. Later on, bismuth combined with opium is the best treatment. The milk diet should be supplemented only very gradually.

#### CHRONIC DISORDERS OF THE STOMACH.

§ 204. *The patient, whose temperature is normal, complains of "Chronic Indigestion," i.e., pain or discomfort in some way connected with his food, which has probably come on gradually, and may have lasted a long time. There are SIX DISORDERS from any*

<sup>1</sup> One case of this rare condition which recovered is recorded by Dr. Grunbaum in *The Lancet*, Aug. 2, 1902.

<sup>2</sup> The products of the decomposition of nitrogenous food stuffs, especially when enclosed in hermetically sealed tins, occasionally give rise to the formation of toxic substances. The effects are very severe. Symptoms of acute irritant poisoning come on within a short time after the meal; the collapse is extreme, and death may take place within a few hours.

<sup>3</sup> I remember being called to see a medical man, about 45 years of age, of markedly gouty diathesis, who had previously had gouty manifestations. The symptoms were thought at first to resemble those of enteric fever, the temperature being 103° to 105° for several days; but a brisk purge of calomel, salicylates, alkalies and milk diet produced immediate improvement.

one of which he may be suffering, and there may be *Dilatation of the Stomach* in addition<sup>1</sup>—

a. **Functional** diseases of the stomach **without tenderness.**

I. Atonic Dyspepsia.

II. Acid or Irritable Dyspepsia.

III. Gastralgia.

b. **Organic** Diseases of the stomach **with marked local tenderness and pain.**

IV. Simple Ulcer of Stomach.

V. Cancer of Stomach.

VI. Chronic Gastritis.

c. There are also many other disorders **unconnected with the stomach** which may give rise to symptoms of chronic indigestion, among which the following may be mentioned:—Phthisis (of which dyspepsia is often the earliest symptom). Appendicitis, Abdominal tumour. Cardiac or Hepatic disease. Renal or Uterine disease, various Nervous disorders, and Pancreatic disease (rare).

I. *The patient complains of CHRONIC INDIGESTION, and the epigastric pain or discomfort comes on SOON AFTER A MEAL*—the disease is probably **ATONIC DYSPEPSIA**.

§ 205. **Chronic Dyspepsia** may be defined as deranged digestion without gross or inflammatory changes in the mucous membrane of the stomach. It may be, and often is, attended by Atony or Dilatation, § 211. It occurs in two generally accepted types.

<sup>1</sup> As I have already remarked, this classification is a very unsatisfactory one. As more scientific methods of investigation, such as those foreshadowed in §§ 199 and 200, come to be employed, we shall be able to classify cases which we now vaguely describe as "CHRONIC DYSPEPSIA" into the following groups—groups which tell us wherein the chief error of digestion lies.

I. **DISORDERS OF SECRETION.**

a. *Irritative states*—1. Hyperacidity (Hyperchylia), Hyperchlorhydria, excessive formation of HCl. 2. Supersecretion (gastrosuccorrhœa), a continuous flow of gastric juice: doubtful if this exists apart from dilatation.

b. *Depressive states*—1. Subacidity (Hypochoylia). 2. Inacidity (Achyia gastrica).

II. **DISORDERS OF MOTILITY, or Peristalsis.**

a. *Irritative states*—1. Cramp of the cardia, pylorus, entire stomach. 2. "Peristaltic unrest" of Kaussmaul. 3. Nervous eructations. 4. Nervous vomiting.

b. *Depressive states*—1. Insufficiency of the cardia or the pylorus. 2. Gastroplegia—atony or insufficiency of the entire gastric muscle leading to dilated stomach.

III. **SENSORY DISORDERS.**

a. *Irritative states*—1. Hyperæsthesia. 2. Gastralgia. 3. Bulimia and polyphagia.

b. *Depressive states*—1. Anorexia. 2. Acoria (? gastric anæsthesia).

IV. **GASTRIC NEURASTHENIA; and GASTROPTOSIS.**

These various conditions are of course met with most frequently in combination, just as paralysis and anæsthesia are met with in disease of the spinal cord. Thus atony and dilatation inevitably lead to disordered secretion. But it is of the greatest use for purposes of prognosis and treatment to know which particular element in the digestive process is at fault. The subject, however, is extremely complex; for instance, the gastric contents, in cases of Subacidity, may be highly acid from the presence of fatty acids the products of decomposition. Those desirous of pursuing this subject should consult Henmieter's "*Diseases of the Stomach*" (Blakiston: Philadelphia), or the works of Mathieu (*Traité des Maladies de l'Estomac et de l'Intestin*, Paris, 1901), Ewald, Reissmann, Einhorn, and Rosenheim.



I. ATONIC DYSPEPSIA (the commoner form) is chronic indigestion due to diminished digestive power of the stomach. It is probably due to a deficiency of the acid in the gastric juice; the pepsin is said by most to remain normal in amount. In this disease the food may, in process of time, undergo butyric acid fermentation, and then it is difficult to distinguish this form of dyspepsia from II.

II. ACID or irritable DYSPEPSIA (§ 206; *Synon.*—hyperchlorhydria) is a chronic indigestion due to hypersecretion of acid in the gastric juice.

I. **Atonic Dyspepsia** is the commoner form of chronic dyspepsia. The *symptoms* are:—(1) Pain or distress, usually in the epigastrium, coming on immediately or *very shortly* after food. The pain may be in the back or shoot up to the shoulders; or there may be no definite pain, only a feeling of weight or distension. It is unaccompanied by tenderness on pressure, a feature which distinguishes it from gastritis and other organic conditions. The pain is often relieved by eructations of wind. (2) Nausea and vomiting are not frequent. (3) The appetite is usually diminished, but sometimes it is increased; and the tongue is flabby and indented by the teeth. (4) There are languor, depression, general discomfort; and there may be palpitation, dyspnoea, and other cardiac symptoms. Thirst is not usual unless there be dilatation, and pyrexia is absent. Urates in excess are constantly present in the urine.

*Etiology.* (1) Errors of diet (see § 214). (2) Overwork, mental anxiety, and other nervous derangements: reading during meals is said to give rise to it. (3) Imperfect mastication in previous years. (4) Convalescence from acute diseases, anaemia, and debility from any cause (*e.g.* phthisis), predispose. Dyspepsia is often the earliest symptom met with in phthisis. (5) Various abdominal disorders, *e.g.*, pancreatic or renal disease, appendicitis, and abdominal tumour, may for some time be evidenced only by symptoms resembling atonic dyspepsia.

*Diagnosis.* The chief condition from which it has to be distinguished is *chronic gastritis*, in which there is usually tenderness on pressure; and, while stimulating articles of food (pickles, condiments, etc.), relieve the pain of atonic dyspepsia, they tend

to aggravate chronic gastritis (see also Table XVII.). Atonic dyspepsia may have to be differentiated from *gastric ulcer* in the young, or *cancer of the stomach* in the middle-aged and old (*q.v.*). The differentiation from *acid dyspepsia* is given under that disease (§ 206), but it must be remembered that in atonic dyspepsia butyric acid fermentation is apt to take place, and it is then almost indistinguishable from acid dyspepsia, excepting by an examination of the stomach contents.

TABLE XVII.

	CHRONIC DYSPEPSIA.	CHRONIC GASTRITIS.
<i>Tenderness</i> .	Absent.	Present.
<i>Vomiting</i> .	Not frequent, but relieves pain.	Frequent, especially in the morning, of mucus: no relief.
<i>Thirst</i> . .	Varies; not common.	Usually marked.
<i>Fever</i> . .	Absent.	Sometimes slight fever.
<i>Causes</i> . .	1. Dietetic errors. 2. General weakness of system (anæmia after fevers, etc.); or nervous exhaustion, leading to <i>deficient secretion</i> of gastric juice.	1. Dietetic errors, especially alcoholic excesses. 2. Sequel to Heart or Liver disease.
<i>Course</i> . .	Liable to come on in attacks lasting a few days or weeks at a time; brought on by slight causes.	Does not come and go, but progressively advances, and goes on to dilatation of the stomach.

*Prognosis.* It is never fatal, but often makes the life of the patient very wretched, and unfits him for the duties of life. If met with early, treatment may be very efficacious; but, if untreated, it may go on to chronic gastritis and dilatation of the stomach, and lead to general malnutrition (§ 195).

*Treatment.* The indications are: (1) To remove the dietetic errors (see § 214). In anæmic cases, soups and stimulating proteid foods are better than farinaceous ones. (2) To stimulate the secretory and motor power of the stomach. This may be done by the use of alkalis and alkaline carbonates shortly before meals, combined with *nux vomica*, bitters, and carminatives (F. 66). Some cases do best by taking their meals dry, so that the gastric juice may be undiluted. (3) Some find aid in pepsin, pancreatin, peptenzyme, taka-diastase, or other artificial digestive. (4) Various symptoms require treatment. For the *flatulence*

20 grs. sodii bicarb. in a cupful of hot water gives great relief. Peppermint, sp. chloroformi, rhubarb, cinnamon, ginger, cardamoms, pepper, charcoal, or F. 50, are all useful. *Acid eructations* (which are generally due to butyric acid fermentation) may be counteracted by antiseptics (carbolic acid or creosote, etc.), or acids after meals. *Pain* disappears when the gastric juice is increased; otherwise bismuth, hydrocyanic acid, and opium (with caution)<sup>1</sup> may be called for. For *breathlessness*, *palpitation*, and other *cardiac symptoms*, sal volatile, saline purgatives, and alkalis may be given. (5) Tonics are useful in the later stages, especially strychnine. For further detail as to treatment see chronic gastritis (§ 210).

II. *The patient complains of* CHRONIC INDIGESTION, *but the discomfort does NOT COME ON DIRECTLY after a meal, and may be relieved by food*—the disease is probably ACID DYSPEPSIA.

§ 206. **Acid Dyspepsia** (Irritable Dyspepsia, Hyperchlorhydria, compare § 200) may be defined as chronic indigestion due to the hypersecretion of hydrochloric acid in the stomach; pepsin, according to most authorities, remaining constant in amount. Opinions are divided as to whether this is or is not a pure neurosis. Some regard it as a *chronic glandular gastritis*, set up by local irritation of injudicious food, alcohol, or the decomposition of retained food residues.

The *symptoms* which distinguish this from Atonic Dyspepsia, which it resembles in other respects, are: (1) Pain, severe, gnawing, intense, burning, *coming on one or two hours after food*; unattended by tenderness on pressure; and usually relieved by taking food. (2) Vomiting may occur, or acid eructations, which may be so acrid as to make the throat sore; thirst; and generally an increased appetite. (3) *The presence of HCl in an empty stomach*, say before breakfast, is the crucial test of hypersecretion.

*Etiology.* (1) It is usually met with in young adults, or men in the prime of life with strong constitutions. (2) Some maintain that this is in reality a neurosis (*vide supra*). (3) It may arise from excess in alcohol, or highly spiced foods, or simply overloading of the stomach.

<sup>1</sup> De Quincey started his habit of "opium eating" for an intractable form of chronic dyspepsia.

*Diagnosis.* Acid may be distinguished from *Atonic Dyspepsia* by the above symptoms. For the diagnosis from *Chronic Gastritis*, see § 210, and Table XVII., p. 362. *Cancer* is detected by the "coffee-ground" appearance of the vomited matters, and the cachexia of the patient. *Gastralgia* may simulate acid dyspepsia; but in the latter the pain is relieved by alkalies, while in gastralgia it is not so relieved. The examination of the stomach contents shows that in hyperchlorhydria the proteids are more completely digested than in gastralgia.

The *Treatment* is mainly dietetic (see § 214). A diet of proteids relieves the condition, but if persisted in too long this further stimulates the secretion of HCl. Antacids, such as large doses of sodii bicarb. or cret. preparat., may be given one to two hours after meals. Lozenges constantly sucked, which induce a considerable amount of alkaline salivary secretion, are useful, especially the bismuth, magnesia and chalk lozenge (B.P.). As a temporary measure, the acidity of the stomach may be diluted by a copious draught of hot water, which relieves the pain and acid eructations. A course of galvanism (see § 211), combined with the proteid diet given below, is very efficacious.

In severe cases of Acid Dyspepsia the following diet may be tried for a few days and relaxed gradually afterwards. The *meals* to consist of meat cakes (as much as desired), and one or two slices of toast (meat cakes are prepared by scraping the fibres either of meat, fish, or poultry, with a blunt knife, leaving behind all the gristle and sinews—add a little salt, press into cakes and fry lightly). Only 4 ozs. of *fluid* to be drunk with each of the meals. As much fluid as desired may be drunk, but not nearer to a meal than one hour before or two hours afterwards. Osler recommends strictly meat diet<sup>1</sup>—3¼ oz. meat minced fine, taken raw with 2 slices stale bread and 1 oz. butter, with 1 glass Apollinaris water thrice daily.

III. *The patient complains of sharp paroxysmal PAIN, having NO DEFINITE RELATION to the taking of food, and careful investigation reveals no structural disorder of the stomach*—the case is probably one of GASTRALGIA.

§ 207. *Gastralgia* is a gastric neuralgia, sometimes attended by a hyperæsthesia of the mucous membrane of the stomach, but always without structural changes or alteration of secretion.

<sup>1</sup> Herschell (*B. M. J.*, 1898, vol. ii., 1323) holds that cases of *Hyperchlorhydria* get worse on the administration of a largely or entirely proteid diet by the encouragement thus given to the hypersecretion of acids. He therefore suggests the substitution of a carbohydrate diet partially dextrinised by taka-diasase, and the neutralisation of the hyperacidity by large doses of alkalies. Weak solutions of tannin are also good.



*Symptoms.* (1) The pain is of a sharp or burning character, in the epigastrium, usually relieved by pressure. There is generally no tenderness, but if present, it is usually more marked with a light than a heavy touch, thus differing from organic disease. Sometimes it is unilateral. The pain may begin immediately after food, but may come on either when the stomach is empty, or when it is full; the *irregularity of its advent* is one of its most characteristic features. Sometimes it comes on with the first mouthful of food; sometimes food relieves it; sometimes it occurs in attacks quite unrelated to food. Dieting gives no relief; for it may be worse after a milk diet than after raw apples. (2) Vomiting and other symptoms are rare. (3) It generally occurs in neurotic people, who have had neuralgia elsewhere. (4) It may accompany gastric ulcer, or follow this and other diseases of the stomach.

*Etiology.* (1) Gastralgia may come on at any age, and in either sex. (2) Some constitutional state, such as hysteria, neurasthenia, anæmia, ague, alcoholism, or gout, is usually present at the same time. (3) In tabes dorsalis gastralgia is the most frequent form of crisis ("crise gastrique").

*Diagnosis.* Those cases of gastralgia in which food relieves the pain have to be diagnosed from *Hyperchlorhydria*. The diagnosis in such cases is effected, first, by administering alkalies an hour or so after meals; they relieve the pain of hyperchlorhydria, but not that of gastralgia. Secondly, acid eructations are a prominent feature of hyperchlorhydria, but not of gastralgia. Thirdly, excess of hydrochloric acid is found on examining the stomach contents in hyperchlorhydria. *Ulcer of the stomach* has a more limited area of tenderness on pressure, the pain comes on immediately after food, and is relieved by vomiting; increased HCl is found on examination of the vomited matter. *Cancer* of the stomach is very difficult to diagnose from gastralgia before tumour or hæmatemesis supervenes; but in this disease the pain is usually more constant. *Biliary colic* is usually associated with jaundice.

*Treatment.* (1) Treatment directed to the constitutional condition generally relieves the gastric trouble sooner or later. (2) Warmth to the epigastrium, opium (with caution), nitrate of silver internally (gr.  $\frac{1}{6}$ ), and arsenic in small and frequent doses. For the vomiting give hydrocyanic acid, bromides, and liq. arsenicalis (℥ i. in a drachm of water every  $\frac{1}{2}$  hour).

GROUP b. If the patient complains of **Chronic Indigestion**, attended by pain and marked **tenderness on pressure**, we are justified in suspecting the presence of organic disease of the stomach, viz., IV. SIMPLE ULCER; V. CANCER; or VI. CHRONIC GASTRITIS.

IV. *The patient is an anæmic young woman, and complains of severe PAIN, PRODUCED BY FOOD and RELIEVED BY VOMITING, the vomit sometimes containing a large quantity of blood*—the disease is SIMPLE ULCER OF THE STOMACH.

§ 208. **Simple** (*i.e.*, non-malignant) **Ulcer** of the stomach, is so called in distinction from cancerous ulceration. The ulcer is usually single, and generally situated on the posterior wall, near

the pylorus on the lesser curvature. In this disease there are, in addition to symptoms of chronic dyspepsia, three very characteristic features:—

(1) *Pain* of an intense boring character usually limited to one spot, (2) *aggravated by food*, and accompanied by tenderness. (3) The pain is *relieved by vomiting*, which comes on very shortly after food. The vomited matter contains an excess of hydrochloric acid. (4) *Hæmatemesis*, which may be profuse, comes on suddenly from time to time. (5) The appetite is usually normal or increased, but the patient avoids food because of the pain it produces. There is generally constipation. In some cases all symptoms may be in abeyance, the disease being quite *latent*, until hæmorrhage or perforation suddenly occurs.

The *diagnosis* is not difficult in presence of the three symptoms above mentioned. These, especially when associated with profuse hæmatemesis, are quite distinctive. When one or other is absent the disease has to be diagnosed from *gastralgia* (§ 178); from *cancer* and *chronic gastritis*, see Table XVIII., below, or from the other causes of Hæmatemesis (§ 193).

TABLE XVIII.

	SIMPLE ULCER.	MALIGNANT DISEASE.	CHRONIC GASTRITIS.
<i>Vomiting :</i>	Frequent; directly after food; relieves pain.	Infrequent; but very large quantity every few days.	Morning vomiting of mucus.
<i>Hæmatemesis :</i>	Occasional but profuse; therefore bright red.	A continuous oozing, therefore "coffee-ground" in character.	Rare; and only streaks, unless in the venous congestion due to heart disease.
<i>Tumour :</i>	None.	Present, though may not be palpable; secondary deposits may be recognisable in liver, peritoneum, glands, etc., later on.	None.
<i>Age :</i>	Young women 20—30.	Usually men over 40.	Any age.
<i>Course :</i>	Indefinite; relapses occur.	Fatal in 1—2 yrs.	Indefinite—may go on to Dilatation.

*Etiology.* Sixty per cent. of Welch's cases<sup>1</sup> occurred in females, and forty per cent. in males. The favourite age in the former was 20—30, in the latter 40—50. It is very apt to occur in association with anæmia, and anæmic states generally (see Chapter XVI.), and some cases have been traced to embolism from heart disease. Some say it is more common among the poorer classes; it certainly is very often met with among domestic servants.

*Prognosis.* On the whole, the mortality is about 10 per cent. The prognosis is usually favourable if the condition is treated early, but there is a great tendency to relapse. If untreated, perforation into the peritoneal cavity may cause death (see § 170). When a more favourable course is followed the resulting cicatrization may lead to distortion or stricture of the stomach or pylorus. Adhesions to surrounding viscera, subphrenic abscess, or abscess in other situations may result. Death occasionally results from hæmorrhage. The amount of the bleeding is no measure of the size or depth of the ulcer; it may be so small as to escape detection post-mortem.

The *treatment* of the hæmorrhage is fully given under Hæmatemesis (§ 193). In the intervals, liq. potassæ, and alkaline carbonates, bismuth, small doses of tannin, and many other of the remedies used in gastritis (*q.v.*) have been recommended. For the pain, give hydrocyanic acid, opium, bismuth, alkalies, or cocaine. In very chronic cases nitrate of silver may be tried. But the chief, and indeed the only successful treatment consists in the regulation of the diet. Two to three pints of milk should be taken daily, never more than a small teacupful at one time. Even bread may cause pain or be rejected by the stomach. Pain and nausea are the symptoms of intolerance. In some intractable cases it may be necessary to feed solely per rectum (*vide* nutrient enema, F. 74). I remember one patient who increased in weight about 1 lb. a week for ten weeks under this treatment, and finally recovered. Solids should only very gradually be renewed, beginning with a small quantity of bread, a lightly boiled egg, then boiled fish, then boiled fowl. To regulate the bowels, the best form of aperient, if enemata fail, is a drachm of Carlsbad salts in

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<sup>1</sup> Quoted by Osler, "Prin. and Prac. Med." (2nd ed.), p. 394.

3 or 4 ozs. of water (120° F.) taken every 15 minutes in 4 doses up to half an hour before breakfast.

**Duodenal Ulcer** occurs mostly in males; Osler refers to statistics where the condition occurred in 171 males and 39 females. The symptoms may be very obscure: sometimes there is only a fixed abdominal pain. It has been diagnosed clinically by three symptoms occurring in a person of the male sex: (1) Sudden intestinal hæmorrhage, preceded or accompanied by hæmatemesis; (2) pain 2 to 3 hours after food in the right hypochondrium; (3) severe attacks of gastric pain from time to time, usually with hæmorrhage.

V. *The patient, who is in middle or advanced life, presents more CACHEXIA than could be accounted for by dyspepsia, and vomits from time to time "COFFEE-GROUND" MATERIAL*—there is probably **MALIGNANT DISEASE OF THE STOMACH.**

§ 209. **Cancer of the stomach.** The stomach is a frequent site for the primary deposit of cancer; it has been found in as many as 1 per cent. of all post-mortems. The word cancer is associated in our minds with a tumour, but in two-thirds (two-fifths Hemmeter) of the cases of cancer of the stomach there is no tumour, but a scirrhus infiltration of the pylorus, which produces obstruction of that orifice and leads to Dilatation (§ 211). The clinical history, which rarely extends beyond one or two years, may be described in three stages. In the first stage we find the symptoms of chronic gastritis (§ 210) combined with marked cachexia. In the second stage, combined with these are acute pain (generally), vomiting, and hæmatemesis of a very characteristic kind. In the third stage, besides the preceding, we get either dilatation of the stomach, or tumour, or both. In many cases, however, there are no symptoms referable to the stomach, and the diagnosis is only made in the deadhouse.

*Symptoms.* (1) *Loss of appetite*, soon followed by *cachexia*, occur early and are very marked; and these symptoms in a patient of 40 or upwards, should always make us suspect the condition. The sallowness of the skin may almost pardonably be mistaken for Addison's disease, or even jaundice. (2) The *pain* is situated in the epigastric region or back, radiates in various directions, and is usually accompanied by tenderness. It is continuous, sometimes increased by food, but sometimes unrelated to the taking of food. (3) *Vomiting* is a fairly constant sign. Generally it takes place some time after the ingestion of food, the interval depending



upon the position of the lesion; thus if at the cardiac end the interval is short, if at the pylorus it may be hours after taking food. Sometimes the vomiting occurs every 2 or 3 days. An examination of the vomited matter shows, according to some, the absence of hydrochloric acid and the presence of lactic acid. (4) *Hæmatemesis* is generally present sooner or later. The bleeding is small in quantity, but occurs frequently, and therefore the blood is partly digested, and gives rise to a characteristic brown appearance, as of *coffee-grounds*. (5) *Dilatation* of the stomach is sure to ensue if the pylorus is involved (§ 211). *Sarcinæ* (Fig. 63) and other evidences of decomposition may be present, and sometimes cancer cells. (6) *Tumour* is much less rarely met with than one would expect. When cancer is deposited in the pylorus it may cause adhesions which prevent the tumour from coming forward. Distension of the stomach by copious draughts of water may help us in the physical examination.<sup>1</sup>

*Etiology.* (1) Cancer of the stomach is more frequent in men. (2) It is rarely met with under 40, although I have seen one case of 28, another of 30, and several between 30 and 40 years.<sup>2</sup> (3) Hereditary influence often exists. (4) Simple ulcer and chronic gastritis appear to predispose.

*Diagnosis.* Anorexia and cachexia are the only constant symptoms. When the typical vomiting is absent the real nature of the case may be readily overlooked. The deficiency of HCl, and the presence of lactic acid, may aid in the detection of cancer, but they must not be solely relied on. If emaciation be rapid, and gastric symptoms resist treatment, cancer should be strongly suspected. (1) *Dyspepsia* and *chronic gastritis* have pain directly related to food; for these, and (2) *Simple ulcer* of the stomach, see Table XVIII., p. 366. (3) *Simple pyloric stricture* (*vide* *Dilatation*). (4) *Tumour of the pylorus* or stomach has to be diagnosed from tumour in the neighbouring regions (§ 189). (5) *Addison's disease* and other cachectic conditions (Chapter XVI.).

<sup>1</sup> The great majority of gastric tumours come forward to the l. of the middle line. It is usually stated that whereas hepatic tumours move, gastric tumours do not generally move with respiration; but this feature, as Hemmeter points out (*loc. cit.*), has many exceptions. One of greater importance is their alternate appearance and disappearance. At first they are extremely mobile, but later on they become fixed owing to adhesions. This is also the reason why perforation is rare. Pulsation is often communicated to them from the aorta.

<sup>2</sup> My experience of this disease at the Paddington Infirmary was perhaps unusually large. The case *et. 28* is recorded in the *Clinical Journal* about 1888 or 1889.

The *Prognosis* is very grave. The duration is rarely longer than 6 to 18 months after the first definite symptoms appear. Death is the invariable result unless surgical measures are adopted. The symptoms upon which one relies most in the diagnosis in these cases, anorexia and emaciation, have always appeared to me to be those which also best measure the longevity of the patient. Death generally takes place by inanition, but almost as often it occurs suddenly by the involvement of important structures, and it would be unwise to assume that because the patient does not waste he will not die soon.

*Treatment.* The indications are to support the strength and relieve the symptoms. The former may be accomplished by easily digestible or predigested food (§ 214). For the latter consult § 211, Dilatation. For the flatulence and pain, creasote and opium, or morphia hypodermically. Condurango, 30 grs. 4 times a day, is said to be a specific in cancer of the stomach.<sup>1</sup> Pylorectomy and gastro-enterostomy are now successfully performed.

VI. *In addition to other symptoms of CHRONIC INDIGESTION, the patient—who has been, perhaps, the subject of chronic alcoholism, or cardio-pulmonary disease—has VOMITING OF MUCUS IN THE MORNING, sometimes streaked with blood—the disease is probably CHRONIC GASTRITIS.*

§ 210. **Chronic Gastritis** may be defined as a form of chronic indigestion due to parenchymatous inflammation (*i.e.*, chiefly of the glands) of the stomach.

*Symptoms.* (1) Pain coming on shortly after food, usually of a dull character, and attended by tenderness on pressure. (2) Mucous vomiting in the morning, or indeed, mucus found in the stomach contents at any time, is a very characteristic feature of chronic gastritis. Streaks of blood are occasionally present. (3) Thirst is also a prominent feature. (4) A slight degree of pyrexia is sometimes present. (5) The appetite is usually good, but the first few mouthfuls of food satisfy. (6) Flatulence, and other symptoms as in *atonic dyspepsia* (§ 205). (7) General symptoms are invariably present—depression, nervousness, anæmia, loss of flesh, sallowness, and other symptoms referable to the causes of the

<sup>1</sup> *Lancet*, vol. i. (1884), p. 812; and vol. ii. (1886), p. 31.

condition (see below). Chronic gastritis may constitute an early phase of cancer, a fact which it is well to remember; the loss of appetite is then very marked.

*Diagnosis.* (1) *Atonic dyspepsia*, which has no tenderness on pressure, and no mucous vomiting in the morning; and (2) *cancer*, are differentiated in Table XVIII., p. 366.

*Etiology.* (1) Persistent dietetic errors, especially *alcoholic excesses*. (2) Venous congestion, arising either from cirrhosis of the liver, or from heart disease. (3) It may be a sequence of repeated attacks of acute gastritis. (4) Constitutional debility, such as that in Bright's disease, gout, etc., may predispose; and so also may (5) Local causes, such as cancer, ulcer, stricture of the pylorus.

The *prognosis* depends a good deal on the cause in operation and the duration of the symptoms. The case is more grave when due to irremovable venous obstruction. If the disease remain long untreated, the stomach becomes dilated, the walls fibrous, and the glands impaired or destroyed. There are 3 stages: First, simple *congestion*, in which the pepsin is normal in amount but the hydrochloric acid is diminished, and lactic and fatty acids are found. The second stage is one of *mucous catarrh*, in which there is a large secretion of mucus, hydrochloric acid is almost completely absent, and very little pepsin is present. In the third stage there is *atrophy* of the mucous membrane. In this stage both hydrochloric acid and pepsin are absent.

*Treatment.* (1) Here again a correct diet is the most important indication (§ 214). Give small quantities of *dry* food at long intervals (6 or more hours). Alcohol and condiments should be stopped; and smoking must be interdicted. (2) The medicinal indications in the first stage and in mild cases are—(i.) to promote the flow of gastric juice and stimulate the stomach power by bitters, gentian, quassia, nux vomica, capsicum, and carminatives; (ii.) stimulate the secretion of the stomach by alkalies and bitters given before meals. (3) Symptomatic treatment—for the pain, bismuth, mag. carb., and opium; for fermentation and acidity, alkalies, 2 or 3 hours after a meal. Mucous vomiting is relieved by draughts of hot water, with alkalies, before breakfast. If the appetite is absent, give carminatives and bitters; if it is too keen

give bismuth and mag. carb. In the latter condition bitters are harmful, as they excite the nerve endings in the stomach. In the later stages the indications are (i.) to replace the absent gastric secretion, which is done by giving pepsin, hydrochloric acid and predigested foods; (ii.) to prevent fermentation, give alkalies and antiseptics along with meals; (iii.) to prevent acidity and eructations, give creasote, carbolic acid, sulphocarbolate of sodium, etc.

c. *The patient presents all the symptoms of CHRONIC INDIGESTION, and on physical examination there is SPLASHING, or the AREA OF THE STOMACH RESONANCE is increased, or there are FOOD RESIDUES before breakfast*—the disease is probably GASTRIC ATONY or DILATATION.

§ 211. Gastric atony and Dilatation of the stomach are conditions which may accompany or succeed many of the preceding disorders. Gastric Atony, the importance of which has been previously referred to (§ 199), is insufficiency of the power of the stomach to empty itself, independently of pyloric obstruction.

a. GASTRIC ATONY (motor insufficiency) may, it appears, exist in three stages or degrees. (a) In simple *loss of tonicity* the stomach is able to empty itself but there is delay, and splashing can be elicited during the period of digestion, which is prolonged; many of these cases are latent and exhibit no symptoms for a considerable time. (β) *Stagnation myasthenia gastrica*, where the stomach cannot empty itself before the next meal though it does so during the night. (γ) *Retention myasthenia gastrica*, or true dilatation, in which the stomach cannot empty itself during the night, and at all times contains food residues, even when examined by the tube before breakfast. The symptoms of gastric atony are—(1) prolonged lassitude after meals with other symptoms of delayed digestion and atonic or irritable dyspepsia (§ 205); (2) “splashing” several hours after a meal (§ 198); and (3), on percussion or ausculto-percussion some hours after a meal there will be an enlarged area of resonance, particularly to the left of the middle line. This test may be aided by the patient taking a draught of some aerated water, or a solution of sodæ bicarb. 53 grs. followed by tartaric acid 45 grs. in solution (which



generates at the body temperature just 1 litre of  $\text{CO}_2$ ), or by inflation of the stomach by a suitable apparatus. (4) The methods mentioned in § 199 are tedious but useful in difficult cases.<sup>1</sup>

b. GASTRIC DILATATION may be a consequence of gastric atony, or due to pyloric obstruction. Its symptoms are—(1) the same as those of gastric atony in a more marked degree; and (2) definite food residues found in the stomach before breakfast, without which one would not be justified in believing that a condition of permanent dilatation existed. In all cases of suspected dilatation the stomach should be examined by the tube in the early morning, after a long fast. This also gives an important clue to the substances in which digestion is defective. (3) Visible peristaltic movements in the epigastric region may sometimes be seen when the dilatation is due to pyloric obstruction. (4) One of the most characteristic symptoms of dilated stomach due to pyloric obstruction is the vomiting, at intervals of two or three days or more, of large quantities of acid frothy material, containing *sarcinæ* (Fig. 63), on which a scum forms on standing. Vomiting may be altogether absent, but if it is present and has these characteristics we may be satisfied that there is dilatation. (5) The remaining symptoms vary with the *cause*, of which there will be a history, or evidence at the time (*infra*). (6) Autotoxic symptoms invariably ensue—marked lassitude, and various other functional nerve symptoms; sometimes urticaria and other eruptions. Tetany is one of the sequelæ in severe cases.

*Etiology.* Dilatation of the stomach may be a consequence of one of two conditions—ATONY OF THE MUSCULAR TISSUE (a and f below), or PYLORIC OBSTRUCTION (causes b, c, d, and e below).

a. Gastric Atony may occur after prolonged overfeeding, “bolting” the food in early life, alcoholism, chronic dyspepsia (and its causes), or chronic gastritis. Anæmia, rheumatism, enteric, influenza and other toxic blood states have also been mentioned; and there is no doubt that states of general debility, such as those associated with phthisis (especially when combined with excessive feeding) and neurasthenic conditions, markedly predispose.

<sup>1</sup> See also an important discussion on Gastric Atony at the Brit. Med. Assoc., 1902, *The Lancet*, Aug. 2, 1902.

b. Obstruction due to a deposit of scirrhus cancer at the pylorus is one of the commonest causes, and it may produce the most pronounced dilatation (§ 209).

c. Pyloric obstruction may also occur from the cicatrisation of a simple ulcer of the stomach. The age and sex of the patient, and her previous history, are characteristic (§ 208).

d. Pyloric obstruction may be due to pressure from without, e.g., enlarged glands in the fissure of the liver, etc.

e. Pyloric obstruction due to a band of adhesion is rare and difficult to diagnose. It can only be recognised by the exclusion of other causes, and the history of inflammation of the peritoneum.

f. Acute Dilatation of the stomach is a rare variety that is often difficult to recognise. It may come on more or less suddenly in early life, or in states of general weakness, with symptoms of collapse, resembling intestinal obstruction. It is a serious condition.

*The diagnosis* of a markedly dilated stomach is not difficult; the chief question is as to its cause. But the diagnosis of simple atony or myasthenia is always problematical unless the stomach tube or chemical tests be employed.

*Prognosis.* It is always a troublesome malady, especially in cases of incurable stricture of the pylorus. Even in atonic dilatation the cure is very tedious, but the prognosis is ultimately good if the disease be diagnosed early, and the cause removable. Malignant stricture is the commonest cause of pyloric obstruction, and unless dealt with surgically is fatal.

*Treatment.* The indications are : (1) To keep the stomach as empty as possible. This may be done by diet No. II., p. 376, or by washing out the stomach.<sup>1</sup> It should be done last thing every night. It is best to use plain water, according to Dr. Herschell. Give concentrated or predigested foods with very little fluid. Never give carbohydrates and animal foods together. Carlsbad salts carry off much of the residue lying in the stomach when taken every half-hour in the early morning until purging ensues (F. 46 or 51). (2) Give tone to the muscular wall by electricity. (3) Promote digestion (*vide* chronic dyspepsia). (4) To prevent fermentation, the symptoms of which are very troublesome, carbolic acid (m 1 to 3), thymol (gr. v.), or sod. sulphocarb. (gr. xx.), given preferably in a tumbler of water between meals. After

Method, see § 200. Sometimes Turek's double tube is used, the efferent being wider than the afferent tube, to prevent over-distension.

lavage, creasote or calomel ( $\frac{1}{6}$  gr. t. d.) may be given with advantage. Surgical treatment may be needed in cases due to pyloric obstruction, and various forms of pylorotomy have been successfully performed.<sup>1</sup>

Electricity is of great use in dilated stomach, not only for giving tone to the muscular wall, but also for promoting digestion and general nutrition. Faradism may be used, preferably with Einhorn's intra-gastric electrode, but the author has obtained very good results by means of galvanism applied externally.

§ 212. **Neurasthenic dyspepsia** (synon. Gastric Neurasthenia<sup>2</sup>) is probably a form of Gastric atony. We have seen that the nervous system may be seriously deranged as a consequence of gastric disorder, and the opinion is rapidly gaining ground that the motor, and probably the secretory, powers of the stomach may fail as a consequence of functional nervous disorder. The symptoms do not differ materially from Chronic Atonic Dyspepsia due to other causes, as far as our present knowledge goes. Such cases must be recognised by the circumstances under which they occur. Electricity is especially useful in their treatment.

§ 213. **Gastroptosis** (dropping of the stomach: enteroptosis), a condition in which the stomach has dropped from its position, has attracted some attention lately, though considerable doubt still appears to exist in the minds of the profession.<sup>3</sup> The symptoms, and signs also, are apt to be confused with Gastric Dilatation, and indeed it is only with difficulty that they can be distinguished, if at all. An aggravated state of neurasthenia has been usually associated with the condition.

### Dietaries and Invalid Foods.

§ 214. Less food is required in old age than in youth, and with a sedentary life than with an active or outdoor one. For a person in health three meals a day are usually sufficient; but when a man is unable, from illness, to take more than a very small quantity at a time, he may require to take more frequent meals. Dietetic errors are a fruitful source of dyspepsia and gastritis. Too frequent meals, habitual overfeeding, and irregularity of the meals will in time derange any stomach. Deficiency of food, and long restriction to the same kind of food, induce dyspepsia by affording no stimulus to excite the secretions; and in this connection it is well to remember that a frequent cause

<sup>1</sup> See Clin. Soc. Trans., 1899-1900. See also an excellent account of the surgery of dilatation of the stomach by Leonard A. Bidwell, *West London Medical Journal*, April, 1900.

<sup>2</sup> The terms Gastric Neurasthenia and Dyspeptic Neurasthenia should be reserved for Neurasthenia of Gastric origin, as explained in the author's Clin. Lects. on Neurasthenia.

<sup>3</sup> The subject is dealt with at some length in the fifth volume of *Virchow's Archives*, and in Glenard's work (*loc. cit.*), and is referred to under Hepatoptosis and Dislocated Kidney.

of failure on the part of the physician to cure dyspepsia is his disregard of this latter fact. It is a good rule to start treatment by cutting down the amount rather than by entirely prohibiting the use of certain articles of diet. Too frequent a use of condiments, spices, tea, and of alcohol especially, lead to chronic gastritis; while dyspepsia is induced by imperfect mastication, bolting of meals, too much fluid with meals, hard mental or physical work immediately after eating, too cold or too hot food, or food which is badly prepared. Excess of tobacco smoking is certainly a cause of dyspepsia. Greasy and fried foods are bad in dyspepsia because the gastric juice cannot penetrate the coating of fat. "Well-made" pastry and other so-called rich foods are a source of dyspepsia only when taken with much proteid food. Hyperchlorhydria is induced by constant proteid overfeeding.

Without appropriate dietetic rules our best efforts may fail, especially in the treatment of gastro-intestinal disorders, and other diseases which depend on the proper elaboration and assimilation of food. A few specimen dietaries will therefore be given, culled from various authors, or my own experience. These will serve as a basis for any number of other dietaries.

I. The following table is given as a guide to aid in the drawing up of a diet for mild cases of **atonic dyspepsia** or **chronic gastritis**.  
*Breakfast*.—Boiled sole, whiting, or flounder; or a slice of fat fried bacon, or a soft boiled egg; a slice of dry toast with a little butter, or of brown bread (not new) and butter. *Beverage*.—One cup of cocoa, or of milk and water, sipped after eating. *Luncheon*.—Chicken or game, with bread, and a little tender well-boiled vegetable such as spinach, vegetable marrow, or young French beans. *Beverage*.—Half a tumbler of water, sipped after eating. *Afternoon tea*.—A cup of cocoa, or of weak tea with milk, and a slice of brown bread and butter. *Dinner* (two courses only).—Fish of the kinds allowed for breakfast, with a little mashed potato or potato chips. For sweets and dessert, a plain biscuit will suffice. Or, a slice of any tender meat such as saddle or loin of mutton, or the thick part of an underdone chop, a little mashed or grated potato; a spoonful of any plain milk pudding, to which may be added some stewed fruit. *Beverage*.—Half a tumbler of water, with from one to two tablespoonfuls of spirit, if desired.

Condiments and stimulants are good in atonic dyspepsia, but must be avoided in chronic gastritis, as tending to cause further irritation of the mucous membrane. The patient should abstain from salted and cured meats,<sup>1</sup> tinned foods, sweets, pastry, raw vegetables, cheese.

II. The "**Salisbury**" diet consists essentially of the administration of nitrogenous food only; the meals being taken almost without fluid, but a quantity of hot water being taken between meals. There are

<sup>1</sup> Niemeyer, however, reports the cases of a few patients who voluntarily at times restricted themselves to a diet of salted and preserved meats whenever dyspeptic symptoms arose. Probably the success in these cases was due to the fact that such meats are not readily decomposed,



several principles involved in this treatment. In the first place, it is obviously a marked change from a person's ordinary diet, and the principle of "relativity" is introduced. Secondly, the solid food administered is in a highly concentrated form, and gives the stomach a considerable rest from its functions while the diet is administered. Thirdly, there is the elimination of the farinaceous and bulky substances which readily decompose and produce flatulence and kindred troubles. Fourthly, by reason of the dryness and small bulk of the food, a dilated or atonic stomach is enabled to resume its normal dimensions, much in the same way as bleeding will relieve a distended heart. The details of the diet are comparatively simple. One pound (1 lb.) of lean butcher's meat, chopped or scraped very fine and so as to rid it of its white fibrous tissue, and lightly cooked, is taken per diem, divided into 4 or more meals. Occasionally a little well-toasted or twice baked (Zweibach) bread is allowed also. For a change, half-a-pound of fish may be substituted for an equal quantity of meat. The meals are taken quite dry, or 2 ounces of fluid only; but two hours later, half to two pints of hot water is sipped.

**III. Diet for chronic rheumatism** (after Burnett<sup>1</sup>). All rich, sweet, and complicated dishes should be avoided, and also all cured meats, dried fish, pies and pastry or sweets. *Breakfast*.—Fat bacon or fat ham, brown bread and butter, with a cup of cocoa and milk. *Luncheon*.—Fresh fish with melted butter, or chicken, tripe, or calf's head, with mashed potato and well-boiled green vegetables. *Afternoon Tea*.—A cup of tea or of cocoa with milk, and a slice of thin bread and butter. *Dinner*.—If fish has been taken at luncheon, any plain meat once cooked may be taken at dinner with a little mashed or grated potato and some well-boiled green vegetables; any plain pudding or cream.

**IV. Diet for Obesity** (§ 19). *Breakfast*.—Fish, bacon, beef, or mutton (6 ozs.): one breakfastcupful of tea or coffee without milk or sugar, and one small hard biscuit, or one ounce of dry toast. *Dinner*.—Fresh white fish, beef, mutton, lamb, game or poultry (6 ozs.): green vegetables; one slice of dry toast; cooked fruit without sugar. *Tea*.—A cup of tea without milk or sugar; a biscuit or a rusk; two to three ounces of cooked fruit. *Supper*.—Meat or fish (about 3 ozs.) with toast. If desired, a glass or two of sherry or claret may be taken.

**V. Dietary for Diabetes Mellitus** (Sir William Roberts).

*Allow*—Butcher's meat, poultry, game, and fish; cheese, eggs, butter, fat, and oil: broths, soups, and jellies made without meal and sugar; cabbage, endive, spinach, broccoli, Brussels sprouts, lettuce, spring onions, water-cress, mustard-and-cress, celery. For bread is substituted—bran cake, gluten bread (and meal), almond meal, rusks, and biscuits; also, "torrified" or charred bread; dry sherry, claret, light bitter ale, brandy, and whisky in small quantities; tea, coffee (without sugar), chocolate (made with gluten meal), soda-water, bitartrate of potash water.

*Forbid*—All saccharine and farinaceous foods, bread, potatoes, rice, tapioca, sago, arrowroot, macaroni, etc.: turnips, carrots, parsnips, beans, and peas, crabs and lobsters. Liver contains much sugar forming substances, therefore oysters, cockles, and mussels, which contain relatively large livers, are forbidden. All sweet fruits, as apples, pears, plums,

<sup>1</sup> "Foods and Dietaries," by R. W. Burnett, M.D., London, 1892.

gooseberries, currants, grapes, oranges, etc. Port, and all sweet wines; sweet ales and porter; rum and sweetened gin.

**VI. Diet in Chronic Bright's Disease** (Sir Andrew Clark, modified).  
*Breakfast*.—A plate of oatmeal, whole wheaten meal, or hominy porridge, with cream or good milk; bread or toast and butter; cocoa, tea, or coffee, with plenty of milk added. Or, a slice of well-cooked bacon, fish, or fat ham may take the place of porridge. *Luncheon*.—A little fish, with some melted butter, mashed potato, and green vegetable, biscuit or bread and butter. Or, a basin of vegetable soup, a bit of cheese, bread, butter, and salad. Or, a milk pudding with stewed fruit and cream, bread and butter. *Afternoon Tea*.—A cup of tea with milk, a slice of thin bread and butter, or rusk. *Dinner*.—Soup, purée of potato, chicken, or rabbit, mashed potato, green vegetables, plain or milk pudding, with stewed fruit. —Or—boiled fish, butter sauce, a plain entrée with vegetables, milk pudding or shape, stewed fruit or blanc-mange, biscuit or bread and butter, a glass of plain or aerated water. —Or—fish, soup, game or poultry, mashed potato, green vegetables, macaroni cheese. *Dessert*.—Ripe fruit. *Beverage*.—A glass of plain or aerated water. *The last thing at night*.—A glass of milk and soda-water.

**VII. Regimen for Lithæmia**, early stages of Cirrhosis, or a tendency to Congestion of the Liver. (1) Average daily exercise equivalent to 6 to 10 miles walking in the open air. (2) The patient may eat bread, fresh vegetables (well boiled), farinaceous puddings, eggs, corn-flour, oatmeal, fish, poultry, butcher's meat in moderation. (3) Everything to be prepared in the plainest manner possible. The patient should avoid all rich, sweet, and greasy dishes, butter, fats, and alcohol in any form.

**VIII. Pre-digested foods** are indicated in dilatation of the stomach, cancer, and advanced cases of chronic gastritis. Benger's *Liquor Pancreaticus* is the usual ferment employed, because the pancreas contains both a proteolytic and a diastasic ferment. *Taka-Diastase* is a valuable aid in the digestion of farinaceous foods. The patient takes it with his food at the commencement of the meal.

1. *Peptonised milk*. A pint of milk is diluted with a quarter of a pint of water, and heated to a temperature of about 140° F. Two teaspoonfuls of *Liq. Pancreaticus*, with gr. xx. sod. bicarb. are mixed with it. The mixture is poured into a covered jug, and the jug is placed in a warm situation in order to keep up the heat. At the end of an hour or an hour and a half the product is raised to the boiling point. It can then be used like ordinary milk. Peptonising powders are now to be obtained.

2. *Peptonised beef-tea*. Half a pound of finely minced lean beef is mixed with a pint of water and gr. xx. of sod. bicarb. This is simmered for an hour. When it has cooled down to a lukewarm temperature a tablespoonful of the *Liq. Pancreaticus* is added. The mixture is then set aside for three hours, and occasionally stirred. At the end of this time the liquid portions are decanted and boiled for a few seconds. 3. *Other foods* can be similarly prepared.

4. *Peptonised nutrient enemata*. The enema may be prepared in the usual way with a mixture of milk and gruel, or milk, gruel, and beef-tea. A dessertspoonful of *Liquor Pancreaticus* is added to it just before administration. Another formula is given in F. 74.

**IX. Tapioca soup with cream**.—Take a pint of white stock and pour into a stewpan. When it comes to the boil, stir in gradually one ounce

of prepared tapioca. Let it simmer slowly by the side of the fire until the tapioca is quite clear. Put the yolks of two eggs into a basin, with two tablespoonfuls of cream. Stir with a wooden spoon, and pour through a strainer into another basin. When the stock is cooled, add it by degrees to the mixture, stirring well all the while, so that the eggs may not curdle. Pour it back into the stewpan, and warm before serving. Add pepper and salt to taste.

X. **Beef-tea.** Cut up a pound of lean beef into pieces the size of dice; put it into a covered jar with two pints of cold water and a pinch of salt. Let it warm gradually and simmer for a couple of hours, care being taken that it *does not boil*.

XI. **Improved Beef-tea.**—Three-quarters of a pound of steak, scraped or passed through a mincing machine, and pounded;  $\frac{3}{4}$  lb. cold water, one piece of sugar, one pinch of salt, one teaspoonful of tapioca, simmered in a "Gourmet Boila" for 3 hours.

XII. **Artificial Proteid Foods.** Beef tea and other meat preparations do not contain the nutritive constituents of meat, or only in small quantity, but contain quantities of extractives which may derange the digestion and impair the action of kidneys. *Peptonised albumen* (or peptonised meat) is better, but it is doubtful if the organism in certain states of prostration can reconstruct peptone into albumin, and the taste of peptone is very bitter and nasty. The *albumoses* are intermediate between albumin and peptone. They are freely soluble, tasteless, and readily absorbed and reconstructed into albumin, produce no disturbance of the digestive organs, and do not irritate the kidneys. *Somatose* is a meat preparation of which the albumin is mainly converted into albumose, and Stevenson and Luff<sup>1</sup> have drawn attention to its great value as a nutriment, stimulant, and restorative in debilitated conditions, even when the presence of albuminuria shows the kidneys are deranged. It is a yellow powder, freely soluble and tasteless. *Plasmon* is another artificial proteid food. It is prepared from milk, and contains casein in a soluble form. It is a nutriment of some value.

XIII. **Milk, egg and brandy.** Scald some new milk, but do not let it boil. Put it into a jug, and the jug into a dish of boiling water. When the surface looks filmy, it is sufficiently done, and should be put away in a cool place in the same vessel. When quite cold, beat up a fresh egg with a fork in a tumbler with a lump of sugar; beat quite to a froth, add a dessertspoonful of brandy, and fill up the tumbler with scalded milk.

XIV. **Chicken panada.** Take the flesh from the breast of a freshly roasted chicken: soak the crumb of a French roll or a few rusks in hot milk, and put this into a clean stewpan, with the meat from the chicken reduced to a smooth pulp by chopping it and pounding it in a mortar; add a little chicken broth or plain water, and stir the panada over the fire for a few minutes.

XV. **Whey.** Into warm milk put a sufficient quantity of rennet to cause curdling, and strain off the liquid, which is then ready for use.

XVI. **White wine whey** (especially good for infants with summer diarrhœa). Half a pint of milk is boiled: as soon as it boils add  $2\frac{1}{2}$  fluid ozs. good sherry; allow the mixture to boil for a few minutes, then leave in a cool place in a basin. When the curd falls to the bottom,

<sup>1</sup> *The Lancet*, September 30, 1890, p. 885.

carefully pour off the whey or strain through muslin. In grave conditions with vomiting give a teaspoonful every 10 minutes; in inflammatory diarrhœa give a tablespoonful every hour.

§ 214a. **Artificial feeding of infants.** GENERAL DIRECTIONS. Feed the child regularly; if necessary, wake it for that purpose. Use a boat-shaped bottle, with a rubber teat on the end. Feed slowly, holding the bottle on the slope until the milk in it is finished. Keep the bottle strictly clean by scalding it both before and after it is used. Mix a fresh portion for every meal. Do not overfeed; 2 pints of the mixture in 24 hours is enough for a child under 6 months. No starchy food should be given to an infant under 6 months, for the pancreatic secretion is not established till then. On no account keep a baby at the breast after it is 9 months old—about 6 months is long enough.

UNDER 1 MONTH—Feed every 2 hours from 5 in the morning to 11 at night. Start with  $\frac{1}{4}$  oz. milk to  $\frac{1}{2}$  oz. water, with a pinch of sugar, and gradually increase to  $1\frac{1}{2}$  ozs. A small teaspoonful of cream may be given with each feed. When the warm mixture has cooled down a teaspoonful of lime-water may be added.

FROM 1 TO 3 MONTHS—Feed every  $2\frac{1}{2}$  hours with quantities gradually increasing up to  $1\frac{1}{2}$  ozs. milk to 2 ozs. water.

FROM 3 TO 6 MONTHS—Feed every 3 hours with 2 ozs. milk to 2 ozs. water, gradually increasing strength to 4 ozs. milk with 3 ozs. water.

FROM 6 TO 9 MONTHS—Feed with 5 meals a day. Milk  $6\frac{1}{2}$  ozs. to 7 ozs. with water 2 ozs. With two of the meals add a tablespoonful of some “infants’” food.

FROM 9 TO 12 MONTHS—The bottle may be gradually left off. Morning and evening 6 ozs. bread and milk sweetened. *Lunch*.—Milk and water, bread and butter. *Dinner*.—2 ozs. farinaceous milk and egg pudding on alternate days; a little broth or beef-tea with bread on other days, or meat gravy.

FROM 12 TO 18 MONTHS—Morning and evening about 6 ozs. bread and milk sweetened, and bread and butter. *Lunch*.—Half pint milk, bread and butter. *Dinner*.—Meat or fish three days a week (about 2 ozs. cut up, scraped, or pounded to a pulp), with bread, vegetables, milk pudding, and milk and water. On alternate days give gravy or broth with bread-crumbs, and milk pudding. *Tea*.—Bread and butter and milk.

FROM 18 MONTHS TO 2 YEARS—In addition to the last named diet, give minced meat or fish on alternate days, with finely chopped greens and potatoes; at teatime, cocoa. Mutton and bacon fat finely chopped, and raw meat juice are to be recommended for delicate children.



## CHAPTER XI.

### THE INTESTINAL CANAL.

THE physiological importance of the intestinal canal is evidenced by the fact that its length is between 25 and 30 feet, along the whole of which absorption may take place; yet the first feature of intestinal disorders which strikes the student is their inaccessibility to examination. Of late years, grounds have been adduced for believing that bacilli make their way through the mucous membrane of the intestine into the lymph spaces beneath, and thence into the circulation, particularly when the mucous membrane is unhealthy, abraded, or ulcerated; thus intestinal sepsis constitutes a danger heretofore but little appreciated.<sup>1</sup> In the future, therefore, the bactériology of the intestinal canal will probably assume considerable importance, and the examination of the stools will take its rightful place.

Another striking feature about diseases of the intestines is the disproportionate amount of prostration which accompanies them—a point to which attention has already been drawn (§ 166). For instance, in a patient who is attacked by a slight but sudden diarrhœa or abdominal pain, the feeling of exhaustion, which in some cases may amount almost to collapse, is out of all proportion to the local mischief. This disproportionate degree of prostration or collapse is especially marked in early life, when “diarrhœa” is, mainly on this account, found to be the principal cause of death in children under 2 years of age. Again, among the acute specific fevers we find that the most fatal collapse and prostration occur in those where the chief lesion is in the intestinal canal—in cholera, dysentery, and enteric fever. These facts are possibly accounted for by the circumstance that the chief centre of the sympathetic system (its “brain,” so to speak) is found within the abdominal cavity, in close anatomical relation with the intestines which it enervates.

<sup>1</sup> Compare §§ 179–171, Peritonitis; and Dr. William Hunter — Pernicious Anæmia, Path. Soc. Trans., 1901–1902, and *The Lancet*, 1900, vol. i., pp. 221, 296, 371, and 1902, vol. i., p. 1467.

## PART A. SYMPTOMATOLOGY.

§ 215. The cardinal symptoms of intestinal disorder are DIARRHŒA, CONSTIPATION, and ABDOMINAL PAIN.

ABDOMINAL PAIN is frequently present, especially in the more acute conditions, but by no means always; and abdominal pain may be due to so many other diseased conditions within the abdominal cavity that it has been considered in Chapter IX. (The Abdomen).

DIARRHŒA is a cardinal symptom of intestinal disorders, and it will be fully discussed in Part C. of this chapter.

The same remarks apply to CONSTIPATION, and in this instance we shall have to distinguish simple constipation from that important surgical emergency, Obstruction of the Bowels. This also will be dealt with in Part C.

The GENERAL OR REMOTE symptoms are sometimes, especially in acute cases, of a very severe character, in view of the profound PROSTRATION which is associated with some intestinal disorders; and to this allusion has already been made. PYREXIA is not usually a marked feature in intestinal diseases (see p. 296). In the more chronic forms of intestinal disease EMACIATION is apt to ensue in course of time. Various NERVOUS DERANGEMENTS of a neurasthenic order are sometimes, as in gastric diseases, associated with disorders of the intestinal canal, consequent partly on malassimilation and intestinal toxæmia, and partly, no doubt, arising in a reflex manner by intestinal irritation. In rare instances these are of a most distressing nature, and in one case which I have seen, that of a medical man, who was unable to obtain relief, they led to suicide. Reflex symptoms of a less troublesome order—*e.g.*, vague pains, itching of the nose, or bad dreams—may be associated with intestinal parasites and some other intestinal conditions.

## PART B. PHYSICAL EXAMINATION.

§ 216. The physical investigation of the intestinal canal can only be accomplished by two means, the EXAMINATION OF THE ABDOMEN and the INVESTIGATION OF THE FÆCES.

The **Examination of the abdomen** is not always easy, but it should never be neglected in suspected intestinal disorders.

PALPATION and PERCUSSION will enable us to make out any generalised swelling or localised tumour. The tenderness which sometimes accompanies intestinal disorders may also be elicited. *Scybala* are often present within the colon, and must not be mistaken for the hard nodules of cancer or other tumour. Their mobility is a very deceptive feature, and the occasional presence of diarrhœa may delude us. Their disappearance after active purgation is the only certain method of diagnosis. The reader is referred to Chapter IX. for further details as to examination of the abdomen.

§ 217. **An Examination of the Stools** is always important, and sometimes absolutely necessary for the diagnosis of intestinal disorders. A great deal of information can also be thus obtained with regard to diseases of the other abdominal viscera. The *faeces* should be examined *first* as to their physical properties—colour, consistence, shape, size, odour and reaction; *secondly*, for undigested food and other substances, such as mucus, gall-stones, or parasites; *thirdly*, for the presence of blood; and, *fourthly*, a microscopic examination is often necessary. It is only rarely that we can rely implicitly on a patient's statement, even as to the colour and appearance of the stools; and, however disagreeable it may be, we should, when thoroughness is desired, examine the *faeces* ourselves.

A small portion of the *faeces* may be brought in a tin box; but it is preferable to see them in bulk, the patient having used a night-stool. He should pass water before going to stool. A large wide-mouthed glass jar, closed at the top by a stopper, is a very convenient receptacle for their preservation. Nothing should be added to the motion until the doctor has examined it. Then carbolic solution (1 in 100) may be added if we wish to detect mucus, or to preserve the stool.

**Physical properties of the Stools.** 1. The COLOUR of the *faeces* is normally dark brown. When there is diarrhœa, the stools, at first, are dark from excess of bile pigments; afterwards they become lighter on account of their dilution by the increased watery exudation and the presence of undigested food. (i.) *Clay-coloured stools* are found in cases of OBSTRUCTIVE jaundice. (ii.) *Streaks of blood* may be present. (iii.) *Tarry stools*, of a dark or black colour, are due to the presence of blood which, entering the alimentary canal *high up* (as in cases of gastric ulcer), has undergone “digestion.” (iv.) *Black faeces* are seen when the patient

is taking iron, bismuth, or manganese internally. (v.) Colourless "*rice-water*" or *milky stools* are met with in cholera, severe dysentery, or severe entero-colitis, due chiefly to the presence of serum. (vi.) In infancy the stools are normally of an *orange-yellow* colour, but in "dyspeptic" diarrhœa or enteritis they are generally *green*.

2. THE CONSISTENCE OF THE STOOLS is normally semi-solid, and THE FORM is that of a rounded cylinder. (i.) When passed in hard, dry, roundish balls they are known as *scybalæ*. These are generally coated with mucus. Sometimes the irritation they cause sets up a false diarrhœa, and there are alternating conditions of diarrhœa and constipation, which can only be cured by a course of aperient medicines. (ii.) In typhoid fever the stools often present the appearance of *pea soup*. The *rice-water* diarrhœa of cholera has just been referred to. (iii.) In cases of stricture of the rectum, *e.g.*, from syphilis or cancer, the stools are *ribbon-like* in shape, and this forms an important diagnostic indication.

3. The ODOUR of the stools, which is due to skatol, does not give us much information. There is a characteristic gangrenous odour in severe ulceration—syphilitic, cancerous, or dysenteric. An ammoniacal odour is never met with in human fæces. If this odour be present it arises only from the presence and decomposition of urine.

4. The REACTION of the stools is normally feebly alkaline when first passed, but they may be acid, a feature which is common in infancy. In the course of a few hours the stools undergo acid fermentation.

VARIOUS SUBSTANCES may be found—

1. UNDIGESTED PARTICLES OF FOOD, if in excess, are indicative of imperfect digestion (gastric or intestinal), and, unless the food has been excessive, denote more especially enteric or pancreatic disease (see also p. 390). In children this feature usually indicates over-feeding. Hard concretions, consisting usually of phosphates and other matters, are sometimes found; and these form the dangerous sources in appendicitis.

2. MUCUS in the fæces is often overlooked unless specially sought for. To discover it satisfactorily *water must be added* to the



faeces, when any mucus present will be seen floating about like small pieces of jelly. The presence of mucus in small amount is of no consequence; it is usual in constipation. When in quantity, and intimately mixed with the faeces, it indicates catarrh of the small intestine. When in isolated masses it signifies the presence of catarrh of the large bowel. In membranous, or mucous colitis, long cylinders of mucus are passed, sometimes without much faeces. These cylinders are generally swarming with *b. coli*, which infest the colon.

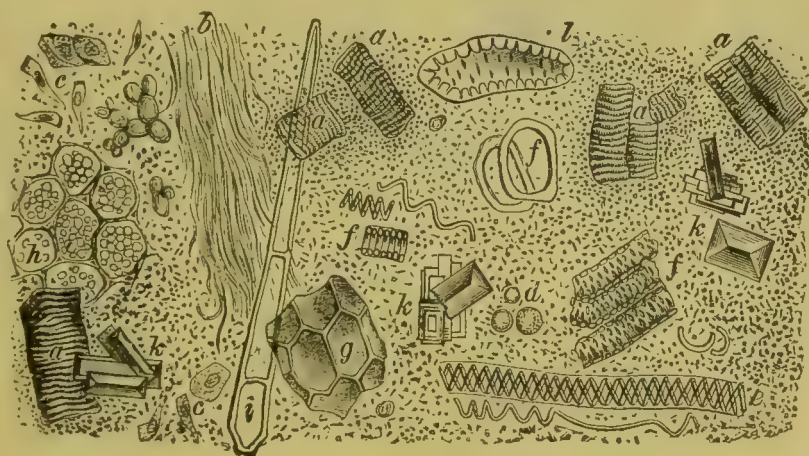


Fig. 64.—MICROSCOPICAL EXAMINATION OF FAECES (after Von Jaksch). Normal appearance  $\times$  about 350. *a*, muscle fibres; *b*, connective tissue; *c*, epithelium; *d*, white blood cells; *e*, spiral vegetable cells; *f* to *i*, various vegetable cells; *k*, triple phosphate crystals in a mass of micro-organisms; *l*, diatoms.

3. BLOOD in the stools may appear either as streaks or in quantity, when from rectum or large bowel. If it comes from the stomach or small intestines it may have undergone partial digestion and give to the stools a tarry appearance (*melæna*). In either case it reddens the water in which the stool is placed, and gives the characteristic spectrum.<sup>1</sup> The causes are dealt with below (§ 226).

4. Pus always indicates *ulceration* of the rectum or colon, which may be of syphilitic, cancerous, tuberculous, or dysenteric origin (§ 223). Pus is difficult to detect when diarrhoea is present. When

<sup>1</sup> Cases have lately been recorded where, after standing for some time, the faeces developed on the exposed surface a colour resembling blood, but no blood was detected by the spectroscope. It appears that in certain as yet unknown conditions some pigment is present in the faeces, which on exposure to the air becomes red like blood. Carter and McManu, *Lancet*, 1899, November 25th, p. 1432.

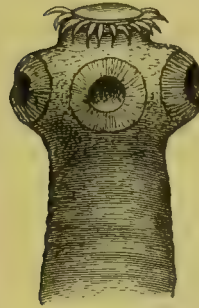
TABLE XIX.—THE PRINCIPAL ENTOMOZOA.

NAME.	CHIEF CHARACTERISTICS OF ANIMAL, AND WHERE FOUND.	CHIEF SYMPTOMS.	OVA OR EMBRYO; CHIEF CHARACTERISTICS, AND WHERE FOUND.	REMARKS.
FAM.: CESTODES.				
<b>Tænia Mediocanellata.</b> (Tapeworm in man.) Fig. 65.	14 to 24 ft. long. Head, 4 suckers, no hooklets. Segments, over 1,000, show central stem uterus with 20 to 30 lateral <i>dichotomous</i> branches. Fastens itself to mucous membrane of intestinal canal in man.	Of reflex irritation, digestive or nervous disorders. Segments passed per rectum.	Recognised by segments containing ova discharged from alimentary canal in faeces. Ova more oval than in <i>T. Solium</i> . Embryo found in beef.	Cattle the intermediate hosts. Found in Great Britain.
<b>Tænia Solium.</b> Fig. 66. (Tapeworm in man.)	About 10 ft. long. Head, 4 suckers, and row of 26 hooklets. Segments, about 850, show central stem uterus with 7 to 10 lateral <i>ramifying</i> branches. Fastens to mucous membrane of intestinal wall in man.	Ditto.	Recognised by segments containing ova discharged per rectum. A six-hooked embryo inside ovum; which eaten by pig, bores its way into the flesh.	Pig the intermediate host—"a measly pork," where scolices abound in flesh. Animal takes 3 months to develop in man. Confined to Great Britain.
<b>Bothriocephalus Latus.</b> Fig. 67.	16 to 25 ft. long. Head club-shaped, with long lateral slits. No hooklets or suckers. About 3,000 segments; uterus, rosette-shaped. Found in intestinal canal of man.	Very few symptoms in adults. Intestinal disorder in children.	Segments containing ova discharged per rectum. Sometimes ova discharged alone; brown shelled; $\frac{1}{16}$ in. long; with a lid at one end.	Ova hatched on reaching water, and eaten by fish, which act as intermediate host. Chiefly found in Switzerland and other parts of Central Europe.
<b>Tænia Echinococcus.</b> (Hydatid cyst in man; tapeworm of dog.) Fig. 75.	$\frac{1}{8}$ in. to 4 in. long. Head pointed, with 4 suckers; double row of hooklets. Has 4 segments, the 4th longer than all others. Found in intestinal canal of dog or wolf.	Hydatid cysts form in liver, or other organs, in man.	Ova found in faeces of dog or wolf. Embryo reaches man by drinking water, and becomes encysted in various organs.	Man is the intermediate host; dog or wolf—the host. Man receives the embryo by drinking contaminated water— <i>e.g.</i> , mountain streams.
FAM.: NEMATODES.				
<b>Oxyuris Vermicularis.</b> (Threadworm.) Fig. 68.	F. = $\frac{1}{2}$ in.; M. = $\frac{1}{4}$ in. in length. Found in large intestine, chiefly the rectum.	Reflex irritation. Worms tend to migrate, at night, and cause itching of anus and genitals.	Worms easily seen after aperients.	Often trouble children. Found in all countries.
<b>Ascaris Lumbricoides.</b> (Round worm.) Fig. 69.	M. about 6 in. long; F. 12 in. Found in small intestine of man.	Reflex irritation, nervous and digestive.	Oval, $\frac{1}{10}$ in. to $\frac{1}{16}$ in. diameter. Hard, dark shell <i>with excrescences</i> . Found in faeces.	Worms tend to migrate, and are sometimes vomited. Frequent in children. Are found in all countries.

(Continued on page 388).



Fig. 65.—HEAD OF *TENIA MEDIOCANNELLATA*. Magnified about 10 times.



a

b

Fig. 66.—*TENIA SOLIUM*. Head  $\times 30$  (a); and proglottides (b) or segments (slightly enlarged). In the latter the uterus has 7 to 10 lateral branches which *ramify*. But in *T. Mediocanellata* there are 20 to 30 lateral branches, which in turn terminate in two branches (dichotomous branching).



b



Fig. 67.—*BOTHRIOCEPHALUS LATUS*. Natural size. Head (a); proglottides (b); and tail (c).



a

Fig. 68.—Female *OXYURIS VERMICULARIS*.  $\times$  about 3 times. a, natural size, about  $\frac{1}{8}$  inch.



Fig. 69.—*ASCARIS LUMBRICOIDES* (Round Worm). About half normal size, and egg  $\times 75$ .

TABLE XIX. (*Continued*).—THE PRINCIPAL ENTOMOZOA.

NAME.	OTHER FAMILIES.	CHIEF CHARACTERISTICS OF ANIMAL, AND WHERE FOUND.	CHIEF SYMPTOMS.	OVA OR EMBRYO; CHIEF CHARACTERISTICS, AND WHERE FOUND.	REMARKS.
<b>Tricocephalus Dispar.</b> (Whip-worm.) Fig. 70.		1½ in. long. Anterior part fine and thread-like. Found in cecum.	Few symptoms.	Ova elliptical, with a projection at each end. Dark coloured, $\frac{1}{100}$ in. long. Found in feces.	Confined to Egypt.
<b>Ankylostomum Duodenale.</b> Fig. 71.		F. = $\frac{1}{10}$ in.; M. = $\frac{1}{4}$ in. Tail end broadest; mouth capsule distinct and provided with 4 tentacles, which clasp the villi of the sm. intestine. Found in jejunum.	Melena, profound anaemia and weakness.	Ova about $\frac{1}{100}$ in. Clear and transparent shell, showing yolks through. Worms found in feces.	Found in Egypt, Brazil, Jamaica.
<b>Filaria Sanguinis Hominis.</b> (Filaria Bancrofti).		3 or 4 in. long; fine and hair-like, lying in bunches together. Found in lymphatics.	Chyluria.	Embryos $\frac{1}{2}$ in. long, enclosed in thin envelope. Tapering tail and round head. Found in blood at night.	Mosquito acts as intermediate host, by taking embryo from blood. Chiefly exist in India and tropics.
<b>Bilharzia hæmatobia.</b> (Distoma hæmatobium.) Fig. 90.		M. = 15 mm. long; cylindrical, with gynephoric canal. F. = 20 mm. Found in blood of portal system, &c.	Hæmaturia and melæna.	Ovary has one terminal spine. Oval, 0.16 mm. long. Found in bladder and urine, and in feces.	Intermediate host probably a fresh-water mollusc. Man gets parasite by water. Egypt, Africa, and Brazil chief countries.
<b>Trichina Spiralis.</b>		M. = $\frac{1}{16}$ in.; F. = $\frac{1}{4}$ in. Lives a few weeks in intestine of man, and then embryos migrate to muscles.	1st stage, gastro-intestinal irritation; 2nd stage, fever, tenderness of muscles, œdema. Often death in 5 weeks.	Larval form bores through alimentary canal, and is found coiled up within a cyst, in muscular tissue. Adult worm and occasionally embryo found in feces.	Man gets parasite from uncooked pig flesh. Rabbits, mice, rats, sheep, also get the parasite. Found in Germany; rare in England.
<b>Filaria Medicinensis.</b> (Guinea worm.)		F. = 1½ in. to 30 in. Cylindrical, white, smooth. Found in subcutaneous tissues of limbs, especially the feet.	Local inflammatory symptoms; later, constitutional symptoms.	Embryo flattened, $\frac{1}{10}$ in. long, with long thin tail.	Presumably acquired by drinking water, which contains intermediate host. Found in Asia, Africa, Egypt, Brazil. Intermediate host—a fresh-water cyclops.



in large quantity, pus indicates an abscess bursting into the bowel, such as a pelvic or ischio-rectal abscess, etc.

5. GALL-STONES may be found by mixing the stools with carbolic water and passing the mixture through muslin or a fine sieve. Gall-stones sink in water when recently passed, though they float when dried. They are very friable, and any suspicious particles should be examined under the microscope for cholesterin, see p. 437.

6. WORMS, such as tapeworms and threadworms, may be found. It is of great importance to find the head of the tapeworm, which

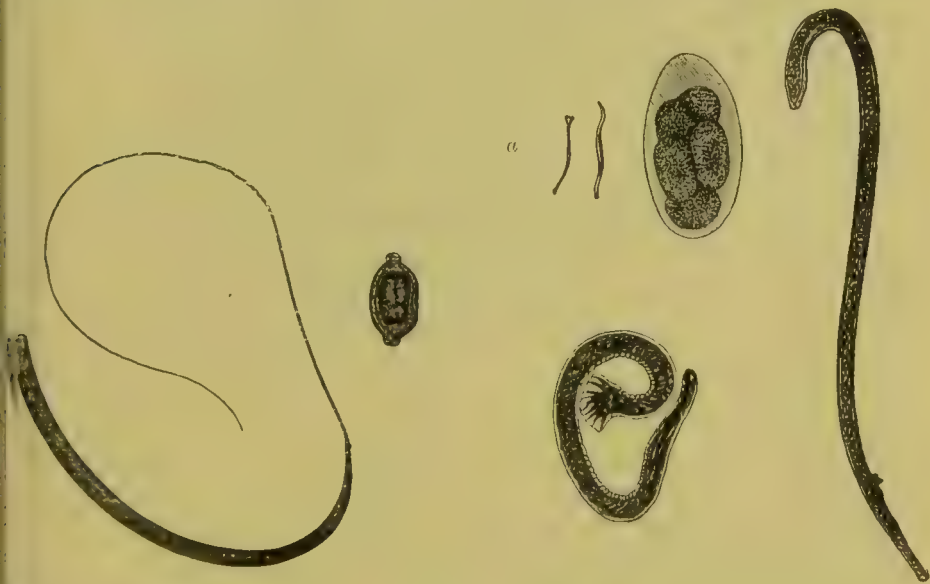


Fig. 70. *TRICOCEPHALUS DISPAR* ("Whip-worm") Magnified by 3, and egg magnified out 100.

Fig. 71.—*ANKLYOSTOMUM DUODENALE* (male (smaller) and female). Worm and egg  $\times 175$ ,  $\sigma$ , natural size.

is about the size of a pin's head. This may be done by the method just described. Another method is, to mix the faeces with water and let the mixture stand. As the parasite sinks to the bottom the supernatant fluid should be carefully poured off, and more added, the process being repeated until the fluid becomes colourless. The various worms are given in the table XIX.

**Microscopic examination** of the faeces is often necessary, especially to find the ova of parasites. Place a small portion of the stool upon a slide, and if not sufficiently fluid, dilute with a quantity of normal saline solution; cover and examine. If dysentery is suspected the stool must be examined whilst still warm. NORMALLY, under the microscope (Fig. 64), the stool shows undigested particles of food, especially starch granules, muscle fibres, connective tissue and fat cells; crystals of fatty

acids, oxalate of lime and other calcium salts. Hamatoidin, phosphates, cholesterin and Charcot-Leyden crystals are rare. Among the bacteria, the bacillus coli communis, various unnamed bacilli, cocci, and yeast are found. Blood corpuscles and intestinal epithelial cells may occur in small amount.

Among the ABNORMAL constituents which should be looked for are, *first* and chiefly, the presence of the ova or segments of the different entozoa (see below § 217a).

2. Among the *undigested food products* an excess of undigested starch or of muscle fibre indicates disease of the small intestine or pancreas. An excess of fat in the faeces indicates either (i.) deficient bile secretion, or (ii.) disease of the pancreas.

3. The *Charcot-Leyden* crystals are the only abnormal crystals of any importance. They are very rare, and are found chiefly in association with worms, especially ankylostomum. Their presence is a useful indication that the parasite is still alive in the intestinal canal.

4. Various *bacilli*, such as those of typhoid and cholera, are present in the faeces in disease, but on account of the many extraneous microbes it is almost impossible to obtain specific cultures from the stools. The *b. coli* has its normal habitat in the colon.

5. The *amœba* of dysentery is more characteristic, and is now regarded as the pathognomonic feature of true dysentery. It was first found in the stools and described by Lösch in 1875, and termed by him the *amœba coli*. They are generally found in fair abundance as roundish cell-like bodies of irregular oval form, which continually undergo amœboid movements. Their size varies from 10 to 20  $\mu$ . Koch was the first to suspect a definite relationship between dysentery and these organisms. Cunningham claims to have found them in cholera patients in Calcutta; and they are now generally believed to be the cause of hepatic abscess.

§ 217a. **Various intestinal and other parasites**, or segments of them, or their ova, may be found in the faeces. These are described in Table XIX. and the accompanying illustrations. Seven of these *infest the alimentary canal of man*—the two common tapeworms (*T. Solium* and *T. Mediocanellata*) recognised by their segments in the faeces, naked eye; the tapeworm of central Europe (*Bothriocephalus Latus*) recognised by its segments, naked eye; the extremely common thread-worm (*Oxyuris Vermicularis*), and the common round worm (*Ascaris Lumbricoides*), both of which may be seen naked eye, the former like small pieces of cotton, the latter as large as a garden worm; and, two worms which are found mainly in Egypt, the *Ankylostomum* and the *Tricocephalus*, both of which with their ova need magnification for discovery. The symptoms and treatment of the commoner of these are given in § 228. The eggs of the *Bilharzia* are sometimes found in the faeces, enclosed in small fleshy masses, but they are chiefly found in the urine, associated with *hematuria*, § 301. The ova are quite peculiar in having a spine-like projection at one end only (Fig. 90). Brief reference may here be made to the four other parasites, though they are not found in the intestines or faeces in man. Hydatid cyst, the cystic (alternate) form of the *T. Echinococcus*, is found chiefly in the liver in man. *Filaria* gives rise to chyluria and is only found in the blood at night. *Trichina Spiralis* is found in the muscles in pork-eating countries. *Filaria Medinensis* is found in the skin in tropical countries.

PART C.—DISEASES OF THE INTESTINAL CANAL, THEIR  
DIAGNOSIS, PROGNOSIS, AND TREATMENT.

§ 218. **Routine, procedure and classification.** Having, firstly, ascertained that the patient's LEADING SYMPTOM is referable to the intestinal canal; and secondly, by inquiries into the HISTORY of the illness, whether it came on *acutely* and suddenly, or gradually in a *chronic* manner; we proceed, in the third place, to the PHYSICAL EXAMINATION of the abdomen after the manner set forth in (§ 167) chapter IX. If, in the course of these inquiries, definite disease can be located in any particular organ, reference should afterwards be made to the appropriate chapter.

A. If **Diarrhœa** is the leading symptom :—

If *acute*, or attended by choleraic or dysenteric symptoms . . . . . turn to §§ 220–2

If *chronic* . . . . . „ § 223

B. If there is **Tenesmus** without diarrhœa . . . . . „ § 225

C. If **Blood** or some other **alteration in the stools** is the leading feature . . . . . „ §§ 226–8

D. If **Constipation** is the leading symptom . . . . . „ § 229

E. If the **Stoppage in the bowels** is complete . . . . . „ § 230

§ 219. **Diarrhœa** is the frequent occurrence of loose or liquid motions; it is the watery consistence of the stools which is the chief characteristic in diarrhœa. A frequent call to stool may arise from some local irritation (see Tenesmus), without any alteration in the consistence or form of the stool. This source of fallacy should be carefully guarded against. Many women speak of the tenesmus which often accompanies the menses as “diarrhœa.”

CAUSES OF DIARRHŒA.

Acute.		Chronic.	
RARE.	COMMON.	COMMON.	I. Acute causes becoming chronic.
			II. Local conditions about anus.
			III. Ulceration (Tuberculous, Syphilitic, Cancer of the bowel, and Colitis).
			IV. Portal obstruction or congestion.
			V. Dysenteric diarrhœa.
			VI. Nervous diarrhœa.
			VII. Amyloid disease.
			VIII. Senile diarrhœa.
			IX. Chronic catarrhal enteritis.
RARE.	COMMON.	RARE.	X. Mineral poisons ( <i>e.g.</i> , arsenic).
			XI. Pancreatic disease.
			XII. Psilosis.

The feces should always be examined where it is possible (§ 217). And sometimes the situation of the disease may thus be discovered. When the stools are coloured with bile, and contain

undigested food, and *small pieces of mucus intimately mixed* with the feces, catarrh of the small intestine may be suspected. When mucus or "slime" occurs in *larger masses*, in "strings" or "casts," there is probably disease of the large intestine.

§ 220. In **Acute Diarrhœa** there is usually a good deal of pain and tenesmus (straining at stool); the tongue is mostly furred, there is thirst, and may be vomiting. If there be much vomiting and prostration the diarrhœa is probably due either to the presence of some violent irritant, or to some serious organic lesion, such as injury to the bowel or peritoneum. In profuse diarrhœa the temperature is usually subnormal, and the urine diminished. It should be borne in mind that scybala retained in the intestines may give rise to attacks of diarrhœa alternating with constipation.

*Causes.* I. The **food** taken, and the vessels in which it has been contained and cooked, should be the first questions to inquire about in all cases of acute diarrhœa coming on suddenly in a healthy person. Collapse and many of the symptoms of cholera can be produced by food cooked in a new copper vessel. One of the irritant poisons may have been introduced into the food accidentally or designedly. This should be borne in mind; and in cases of sudden and unexplained diarrhœa the physician should go patiently through every article taken at every meal during the preceding 24 hours. Over-ripe or decomposing fruit, too much raw vegetable food, tinned meat—especially that which has been long in store, and has undergone a change resulting in the formation of ptomaines (see footnote, p. 349)—shellfish, and bad cheese are also possible causes. The first or diarrhœal stage of trichinosis comes under this heading, and should be considered in pork-eating countries. In cases of acute diarrhœa in which trichinosis is suspected the worm should be sought in the feces, for in the earlier stages of this disease treatment is so much more efficacious. The diarrhœa which precedes the intestinal obstruction caused by intussusception in children frequently follows a heavy meal of indigestible articles; and diarrhœa is itself a cause of intussusception.

II. The quality of the **water** is often responsible for diarrhœa, acute or chronic. This is frequently the case in malarial districts in the summer and autumn, especially when the temperature is



high. Water containing much peat from the mountains may also cause diarrhœa ; and thus the water supply of the town of Montreal frequently occasions diarrhœa in newcomers.

III. **Worms** may give rise to diarrhœa in children. They may be attended by uneasy abdominal sensations, night terrors, picking of the nose, itching of the anus, but sometimes the worms are discovered in the stools when there have been no symptoms pointing to their existence (§ 228).

IV. **Infantile Diarrhœa** occurs in at least three well-recognised clinical forms: (i.) Acute Dyspeptic Diarrhœa, (ii.) Inflammatory Diarrhœa or Entero-colitis, and (iii.) Epidemic Diarrhœa or "summer diarrhœa" (including Infantile Cholera)—mentioned in progressive order of severity.

(i.) In ACUTE DYSPEPTIC DIARRHŒA the stools are offensive, frothy, of a greenish colour, and mixed with curds of undigested food. Vomiting may or may not be present. It is usually a transient condition if adequately treated.

(ii.) In INFANTILE INFLAMMATORY DIARRHŒA (Entero-Colitis) the stools are green, slimy, and often contain blood ; there is some fever at the beginning, and abdominal distension. The inflammation attacks chiefly the colon, consequently there is tenderness on pressure over the region of the colon, and mucus in the stools. Adults also are sometimes affected. It lasts only 1 to 3 weeks if treated as described below.

(iii.) EPIDEMIC DIARRHŒA ("summer" or "autumnal" diarrhœa of children) is met with chiefly in childhood and infancy, in the autumn months of the year, and is attended by catarrh of the mucous membrane of the bowel. The *symptoms* of a severe attack are:—watery stools, foul-smelling, of altered colour, containing lumps of mucus ; vomiting ; acute abdominal pain and tenesmus ; prostration, collapse, subnormal temperature, with pinched aspect, rapid wasting, and often (after a course of a week or so) death from exhaustion. INFANTILE CHOLERA forms about 2 per cent. of "summer diarrhœa" cases. The stools are serous, persistent vomiting is a marked feature ; great collapse rapidly supervenes, the temperature in the rectum is raised as in adult cholera, and death rapidly supervenes : some describe it as a separate affection, but it is probably a severe form of Epidemic Diarrhœa.

*Etiology of Infantile Diarrhœa.*—Any of the previously mentioned causes (I. to III.) are contributory, and very often exciting, causes in all forms of infantile diarrhœa; and especially dietetic errors. This latter is the sole cause in variety i., and probably in variety ii. These diseases affect chiefly hand-fed children, in warm weather, being probably in part due to dirty feeding-bottles, teats, sour milk, etc. Most of the cases occur in children under 6 months old. The causes of *Epidemic Diarrhœa* are far more obscure.—(i.) Seasonal, epidemic, and microbic causes<sup>1</sup> have long been suspected on account of its prevalence during the summer and autumn months. It occurs chiefly after hot dry summers, and the researches of Ballard seem to point to its being dependent upon some telluric condition. (ii.) It occurs chiefly in towns; and certain *localities*, e.g., Leicester, are notorious for a lethal epidemic each summer and autumn. Ballard found that the severity of the annual outbreak seemed to vary with the subsoil temperature; it started when the 4 feet earth thermometer read 56° F. Adults do not altogether escape the influence of these causes, and diarrhœa is widely prevalent in the hot dry summer months in some years; but in children the death-rate is sometimes appalling.

In the *treatment of Infantile Diarrhœa* astringents are not only useless, but harmful. Equal parts of lime water and castor oil (F. 64), every 2 or 3 hours until the stools become healthy, is a most valuable prescription. This must be combined with appropriate diet. The milk must be sterilised, and diluted with lime-water. Where cows' milk cannot be retained, condensed milk, whey, or raw meat juice may be tried. The vomiting may be checked by giving only barley water with white wine whey (§ 214) for a time, and the usual stomach sedatives. In mild cases castor oil, followed by bismuth, rhubarb, soda, and cinnamon, or small doses of hyd. c. cret., or thymol, will effect a cure. But in cases with collapse, brandy is to be given, and the child should be put into a warm mustard bath or hot pack until the skin which is harsh and dry becomes soft and elastic. If much fever is present a cold pack or bath is indicated. For choleraic diarrhœa

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<sup>1</sup> Waldo considers epidemic diarrhœa to be due to local rather than to climatic conditions. Milroy lectures, *Lancet*, May, 1900.

calcium chloride is useful to check the serous exudation, by increasing the coagulability of the blood.

V. **Toxic blood states**, such as enteric fever, are nearly always—whilst measles, and the other eruptive fevers (especially at their advent), Graves' disease, chronic renal disease, uræmia, and pyæmia are sometimes—attended by diarrhœa; and it may also be one of the effects of dissecting-room poison. Gouty people are often subject to attacks of diarrhœa, which, by the way, are of a conservative nature.

VI. A **chill** to the surface in some individuals will determine an attack of acute diarrhœa.

VII. In cases of acute diarrhœa in which the cause is obscure, reference should be made to the **Causes of Chronic Diarrhœa**, any of which may from time to time give rise to an acute attack. **Dysentery** (§ 221) and **Cholera** (§ 222) are the commonest causes of diarrhœa in tropical climates, occasionally in ours.

*Prognosis of Acute Diarrhœa.* The causes of acute diarrhœa are for the most part removable; and though weakened by the attack, the patient generally makes a good recovery. Acute Epidemic Diarrhœa in children, however, is a most fatal affection, and it leads to a higher death-rate in infancy, in Great Britain, than any other disease, accounting for nearly 3,000 *deaths annually in London alone*. Briefly, the prognosis in any given case depends upon (i.) the cause in operation, (ii.) the severity of the symptoms and the evidences of weakness, (iii.) the state of the patient's hygienic surroundings, and (iv.) the effects of treatment. Infantile cholera is rarely recovered from. Dyspeptic diarrhœa may be cured in a few weeks, but if untreated is apt to go on to sub-acute enteritis. Without treatment all forms of epidemic diarrhœa, even in adults, are serious. Should symptoms of prostration or collapse ensue, the outlook is bad; but it is only at the two extremes of life that this disease is so grave.

*Treatment of Acute Diarrhœa.* The indications are (a) to remove any irritating matters present in the intestinal canal; (b) to ensure rest to the irritated parts; and (c) to check excessive exudation. (a) Thus, a simple acute diarrhœa in an *adult* following the ingestion of bad food is cured readily by a dose of castor oil ( $\frac{1}{2}$  oz.) with tr. opii. (℥ x.); or calomel. (b) Milk and bland

food only can be taken ; soups and beef-tea are not advisable. In severe cases withstanding treatment, the diet may be restricted to raw meat juice. Simple cases of dyspeptic diarrhœa in children are readily cured by grey powder every night, and alkaline carbonates during the day. If the stools are slimy bismuth is needed. (c) After the expulsion of all irritant matters, a mild astringent, such as chalk or Dover's powder, is beneficial ; and bismuth to soothe the congested mucous membrane. Astringents are contraindicated in the *early stages* of diarrhœa, especially when due to (i.) irritants, (ii.) inflammation, or (iii.) portal obstruction. Only when the diarrhœa threatens to become chronic, do we require to use astringents, such as catechu, kino, pulv. cretæ. aromat., mineral acids, hæmatoxylin, and tannin. Opium allays irritation and checks peristalsis ; it may be given as tr. opii., tr. chlorof. & morph., etc. Coto is a useful drug ; it acts by diminishing the intestinal secretion. If the stools are very offensive, calomel, charcoal, carbolic acid, and creasote are useful ; and a course of intestinal antiseptics may be given—salol (gr. x.),  $\beta$ -naphthol (gr. v.), thymol, etc. Lastly, when other means fail, rectal injections must be resorted to—opium with starch, or silver nitrate ; but these are useful chiefly when the disease is in the larger bowel. In all severe cases, absolute rest must be insisted upon, with warmth to the abdomen.

*The patient, who is or has been living abroad, complains of severe* DIARRHŒA WITH BLOOD, MUCUS, and perhaps PUS in the stools—the disease is probably DYSENTERY.

§ 221. VIII. **Dysentery** is a form of diarrhœa which occurs in marshy and malarial districts attended by severe tenesmus and frequent stools, and generally with pyrexia, *due to ulceration of the large bowel*, and depending on the presence of one or more specific organisms. It is met with clinically in two forms—(a) *acute* and (b) *chronic*. Both are characterised by (i.) diarrhœa, (ii.) the passage of blood, and (iii.) of mucus, from the bowel.

(a) **ACUTE DYSENTERY** may be of sudden onset : the patient awakens in the early morning with griping pain, and tenesmus, and during the day there may be from 10 to 60 scanty discharges from the bowel, containing blood, mucus, epithelial cells, and later on they acquire the appearance known as the "toad-spawn" discharge. In other cases there is abdominal pain and malaise for a few days before the onset of the diarrhœa. A moderate degree of fever may be present : if at all high, suspect the complication of malaria, or liver abscess. In a favourable case, the discharge ceases after a week or ten days.

(b) *Chronic Dysentery* may result from an acute attack, or it may be



chronic from the onset. In the latter form the patient has a gradually increasing diarrhoea, the stools becoming frequent and scanty, with some tenesmus, and the passage of a little blood or flakes of mucus, the symptoms gradually becoming worse. Dysentery affects the rectum, sigmoid flexure, and descending colon. Sometimes the disease extends as far up as the cæcum, and may consist merely of a *catarrhal* state of the mucous membrane. Severe cases lead to *ulcerative colitis*, when shreds of mucous membrane may be passed, and this may result in thickening and cicatricial tissue, and stricture. The most severe variety, however, is the sloughing or *gangrenous* form, when large sloughs come away with an offensive odour, and are liable to set up septicæmia, or to cause perforation of the bowel, or even a fatal hæmorrhage.

*Etiology.* Dysentery more often affects men, especially if intemperate. Dysentery being a symptom, not a disease, the cause is not in all cases known. True or Amœbic Dysentery occurs in the tropics, where it is epidemic and endemic, and is due to the presence of the amœba coli (see § 217). It is supposed that it enters the alimentary canal by the drinking water. It is more apt to affect unhealthy persons, and is predisposed to by any disease or abrasion of the alimentary canal, such as occurs after eating unripe fruit, a chill, the abuse of purgatives, and especially constipation. In heart and kidney disease, secondary diphtheritic enteritis may occur, which is known as dysentery, but the description above refers mainly to true dysentery.

*Varieties.* a. *Epidemic* Dysentery is most often found in temperate climates, and does not arise from endemic foci. It is worse in summer and autumn. The mortality varies in different epidemics. It is probably spread by the air, food, or drinking water.

b. *Endemic* Dysentery is found in the tropics, for the most part, during hot weather. In temperate climes it may occur in marshy districts, where the drainage is imperfect, and the soil saturated with organic impurities. It is not contagious from man to man, but spreads probably by the drinking water. It is liable to become epidemic.

c. The *Dysentery of War and Famine* breaks out in unhealthy conditions, and is characterised by (i.) being independent of season, though worse in marshy districts; (ii.) very fatal; and (iii.) apparently contagious, as nurses in hospitals are apt to become affected.

*Diagnosis.* Acute dysentery resembles nothing for which it could be mistaken excepting acute diarrhoea, from which it is differentiated by the stools. But chronic dysentery should never be diagnosed before an examination has been made to exclude rectal cancer, polypus, piles, bilharzia; and indeed any of the other causes of diarrhoea (p. 391). Diarrhoea due to ulceration, occurring in a tropical climate, may be mistaken for chronic dysentery. The only pathognomonic feature is the presence of the amœba coli. The ova of bilharzia hæmatobia may be found in small masses resembling polypi. These on being broken up, and examined by the microscope, show the ova (§ 301); the patient will also have a history of hæmaturia.

*Prognosis.* An attack of acute dysentery in a healthy person may pass off in a week or so; but it requires care in a tropical country to prevent it passing on to chronic dysentery, a condition which is very difficult, often impossible, to cure. The sloughing form is extremely dangerous, being so often complicated with grave conditions, such as local abscesses. With chronic dysentery acute exacerbations frequently occur, and the patient becomes anæmic and greatly debilitated by the constant loss of blood. Complications arising in the course of chronic dysentery are ulcers, with consequent periproctitis, abscesses of the liver, cicatrization with rectal stricture, peritonitis, multiple pyæmic abscesses, and pneumonia.

*Treatment.* The main indication in both acute and chronic dysentery is to give rest to the inflamed part. This in the *acute* form is accomplished by keeping the patient absolutely at rest in bed, with no food except white of egg, barley water, chicken broth, etc., for a day or two. The favourite remedy among English physicians is Ipecacuanha. A small dose of castor oil and laudanum may be given at the outset, and if this does not cut short the disease, ipecacuanha must be administered in doses of 25 to 30 grains at a time. The patient is so apt to vomit the remedy that special precautions must be taken in its administration. A dose of opium is administered, and the ipecacuanha given when drowsiness begins; and no food should be taken for 3 hours before and after the drug is given. The patient must be kept lying down, the head low, and no movement permitted. French physicians advise magnesium sulphate (3i. every hour) till there is no more blood and mucus passed, and the temperature is normal. It rarely requires to be given longer than three days. Hot fomentations are used for the abdominal pain, opium in starch enemata relieve the tenesmus; or the bowel may be washed out with boracic solution.

In chronic dysentery the diet must be non-irritating, but it is not good to keep the patient too long on milk food. Sometimes the patient may recover rapidly on being sent a sea voyage. Constipation must be avoided. Astringents must very rarely be employed; though Bael fruit is advocated by some. Enemata of silver nitrate or sulphate of copper (starting with  $\frac{1}{4}$  gr. to the ounce), about two pints at a time, very slowly injected, are the most useful form of local treatment (see also § 223, V.).

*The patient complains of ACUTE DIARRHŒA coming on very suddenly and attended with severe COLLAPSE, abdominal CRAMPS, and "rice water" stools—the disease is probably CHOLERA.*

§ 222. IX. **Cholera** (synon. Asiatic Cholera) is a disease due to the comma bacillus of Koch, which commences with urgent vomiting, purging, and colourless evacuation, cramps and a tendency to collapse, and which, if not fatal in the first stage, is attended by secondary fever. The period of incubation is usually 3 to 6 days, but it may vary between 1 and 10.

There are three well-marked stages:—

(a) Stage of *evacuation*, which lasts from 2 to 12 hours or longer. The patient is *suddenly* seized with violent vomiting, severe cramp, and profuse diarrhœa. The stools, after the first few, are colourless and opaque, resembling rice water, and containing flakes of columnar epithelium and casts of villi; and the *comma-shaped bacillus* (Chapter XX.). There is severe cramp in the fingers, toes, and abdominal muscles, great exhaustion, small and weak pulse, and coldness of the body.

(b) The *algide stage*, cold stage, or stage of collapse. The patient becomes like a corpse; the surface temperature goes down and the skin becomes a deadly livid hue; the pulse cannot be felt at the wrist. The temperature is most remarkable, for in the rectum it may be as high as 105°, while in the axilla it is only 90°. During this stage the purging ceases, but the vomiting and cramps persist. The mind remains clear. There is a suppression of urine and bile.

(c) Stage of *reaction*. The pulse returns, the temperature rises, the bile reappears, the urine is scanty and deficient in urea. The temperature goes up, and may be attended by typhoid symptoms. The bowels are confined. There may be erythematous, urticarial, and other eruptions upon the skin. This stage is followed by great weakness.

The *Diagnosis* is easy in severe cases on account of its extreme suddenness and the severity of the symptoms. The only condition which resembles it is acute poisoning by arsenic, croton oil, and other irritants. The identification of the bacillus renders the diagnosis certain.

*Etiology.* The disease occurs in great epidemics, but it fortunately has not visited this country, except sporadically, since 1865-6-7. Prior to that date there were epidemics in 1854, 1848, and 1832. In India it is endemic, just as typhoid fever is endemic in London. As regards age none are exempt. The season of the year in which all epidemics in this country have occurred has been the autumn and the end of the summer. A hot dry summer predisposes, but the disease is seen in Russia during the winter. The exciting cause is a specific poison which must be introduced into the alimentary canal. The disease is not communicable excepting by the evacuations from the bowels and stomach (like enteric), and cholera requires the same preventive measures (§ 391 *et seq.*). Fresh evacuations will not give the disease; but only when they are slightly decomposed. They take 3, 4, or 5 days to become infectious. The disease is usually propagated by drinking water which has become contaminated. But it may be conveyed in other ways through *want of cleanliness*. One attack does not give immunity from a second.

*Prognosis.* It is a very serious disorder, and nearly all earlier cases of an epidemic are fatal. The average mortality is 40 to 60 per cent. Patients generally die in the algide stage. If they survive this they generally recover, though relapses may occur. In the reaction stage uræmic coma, hyperpyrexia or the typhoid state may cause death. *Untoward symptoms* are, blood in the evacuations, extreme collapse, extreme cyanosis, and absence of the pulse at wrist. Favourable signs are a perceptible pulse in the algide stage, the early occurrence of reaction, cessation of cramp, secretion of urine, and the occurrence of sleep. The commonest *complications* are pneumonia occurring in the reaction stage, bronchitis, pleurisy, parotitis, bed sores, diphtheritic inflammation of the pharynx, genitals, or bladder, and corneal ulcers.

The *varieties* are two. 1. Choleraic diarrhœa or "cholérine," that is, cases like autumnal diarrhœa occurring during an epidemic of cholera. 2. Dry cholera, that is, where there has been no vomiting or diarrhœa, but all the other symptoms. These cases are rare.

*Treatment.* The best treatment for the *preliminary* diarrhœa is to give a small dose of mild laxative, such as castor oil. This should be followed by astringents and other measures to check the diarrhœa, such as rectal injections of tannin, gr. xx. to two pints warm water; and accompanied by light milk diet. When the *stage of evacuation* supervenes keep the patient warm in bed with hot bottles and external warmth of all kinds. The denuded intestine may be protected by large doses of bismuth. A little stimulant may be given, in the form of camphor, sp. ammon. aromat., or alcohol. For the cramps, give morphia. In the *stage of collapse* medicines are useless, as they are not absorbed into the system. Packing in hot blankets and large subcutaneous injections of normal saline (0.75 per cent. solution of salt) constitute the best treatment. Chloroform inhalations have also been suggested. In the *stage of reaction* slight purging is not harmful. Give copious diluents, and if the urine does not appear give an aperient.

§ 223. **Chronic Diarrhœa.** The term chronic diarrhœa signifies the occurrence of frequent *loose* evacuations, say three or more in the twenty-four hours, extending over a period of weeks, months, or even years (as in Sprue). It is usually, though not necessarily, attended by tenesmus. The stools should be examined (§ 217) whenever the cause is doubtful. In all intractable cases the anus should be carefully inspected. Tenesmus points to the presence of disease of the rectum.

I. Chronic Diarrhœa may be due to some of the same causes as **acute diarrhœa** (*q. v.*). In children tubercular ulceration of the intestine, intussusception, worms, or bad feeding; and in adults, errors in diet, ulceration, and chronic irritant poisoning, should be remembered.

II. **Fissure of the anus**, slight ulcers or abrasions, or even an inflamed pile, may cause a chronic diarrhœa, which baffles investigation for a long time. I well remember a woman, æt. 63, who was a patient in the Paddington Infirmary from March 14th, 1887, to January 6th, 1888. She had 5 to 8 motions a day, quite loose, but otherwise normal, without mucus or blood on any occasion. There was no history of dysentery, syphilis, or tubercle. She was treated for 14 weeks by a great variety of remedies. Then, on inspection of the anus, a slight fissure was discovered. She was put on biniodide of mercury; the number of stools immediately fell to 3, 2, and then 1 per diem, and she was cured in 3 weeks.

III. **Ulceration of some part of the intestinal canal** is perhaps the commonest cause of chronic diarrhœa in England, and it will be well to mention here all the ulcerating lesions which may affect the intestine, in order from above downwards. (1) Simple ulcer of the duodenum is a rare condition which may arise from burns, or from the same causes as simple ulcer of the stomach (§ 208). There may be few or no symptoms till sudden peritonitis or copious hæmorrhage and mælena occur. (2) Ulcer of the lower part of the ileum may be due to tuberculosis or typhoid fever. (3) Ulcer of the cæcum may arise from the pressure of inspissated faeces or some foreign body—*e.g.*, the bristle of a tooth-brush—which has been swallowed. (4) Ulcer of the vermiform appendix may similarly arise from foreign bodies, and



cause typhlitis and perityphlitis (*q.v.*). (5) Ulcer of the rectum is mostly of malignant or syphilitic origin; it is attended by the passage of blood and pus, and stricture may result. (6) Ulcers of the large intestine and rectum occur in the later stages of dysentery. These may contract on healing and produce stricture. (7) Cancer of the bowel may produce ulcer in any part of the bowel, but the most frequent situation is the sigmoid flexure. (8) To these some add catarrhal ulceration.

The commonest causes of ulceration in this country are TUBERCLE, SYPHILIS, and CANCER; in tropical climates DYSENTERY.

1. TUBERCULOSIS of the lungs may be attended by diarrhœa, even without ulceration of the bowel, and in such cases the diarrhœa is considered to be one of the symptoms of the hectic fever in pulmonary tuberculosis, or due to swallowing of the infected sputum. This cause, or tubercular ulceration, is recognised by—(i.) evidences of tuberculosis in the lungs or other part of the body; (ii.) the presence of night sweats and intermittent pyrexia; (iii.) the stools are watery and bilious, and there is rarely any pain. (iv.) Relief is generally effected by quinine and opium internally, combined with appropriate dietary; if these fail, recourse may be had to pernitrate of iron, opium, and lead.

2. In SYPHILITIC ULCERATION of the bowel (i.) the motions often consist largely of pus and blood; (ii.) great pain and tenesmus are usual, combined with (iii.) other evidences and a history of syphilis. (iv.) Opium and antisyphilitic treatment are here of great value to check the diarrhœa.

3. INTESTINAL CANCER presents the following features: (i.) the patient is usually over 45 or 50; and there may be a family history of cancer; (ii.) there may be cancer in the glands or other parts of the body, and there is almost always a history of *emaciation preceding the diarrhœa*; (iii.) paroxysmal abdominal pains are frequent, and if the disease is in the rectum there is great pain and tenesmus on passing a motion, if it be not in the rectum, a tumour can generally be made out through the abdominal wall; (iv.) the stools vary, but very often contain blood in quantity.

IIIA. **Colitis**, inflammation of the colon, is one of the most intractable causes of chronic diarrhœa, alternating in the *mucoous* or *catarrhal* variety (§ 179 and p. 403) with long periods of constipation. It occurs in 2 other forms—*ulcerative colitis* which has for causes those just named, and

*membranous colitis* which is probably due to a persistent microbic infection. In the former, which occurs for the most part in adults, there is pus in the motions and usually a history of one of the causes of ulceration above-named, especially dysentery. The latter may occur at any age and is attended by shreds of membrane in the stools. Irrigation of the rectum and colon has been tried, but the radical cure of such cases consists in the operation of right lumbar colotomy. The artificial anus is kept open for 12 months or more, during which time the cæcum and colon are thoroughly irrigated, and finally the opening may be closed.<sup>1</sup>

IV. **Obstruction in the portal circulation** produces diarrhœa due to the congestion of the intestinal wall. It is recognised by—(i.) a previous history of heart disease, or of intemperance and alcoholic dyspepsia; (ii.) other signs of liver or cardiac disease; (iii.) other evidences of portal obstruction, such as ascites, piles, a large spleen, etc. (§ 235); (iv.) there is little or no pain, and the stools are liquid and dark, occasionally bloody. The *treatment* requires caution because the diarrhœa and hæmorrhage of themselves relieve the condition by diminishing the venous engorgement. (i.) If the diarrhœa has not lasted long, a large dose of calomel will relieve the portal congestion and so cure the diarrhœa. (ii.) Mag. sulph. (gr. xx.) with alum and acid sulph. dil. are recommended; bismuth and opium, with caution, are the most useful for checking the diarrhœa.

V. **Dysenteric Diarrhœa** is a sequel of dysentery, which may perhaps have been contracted abroad many years previously. The laity, seeking a more elegant term, often speak of any form of diarrhœa as “dysentery.” The characteristic symptoms here are: (i.) a previous history of acute dysentery, or a residence in dysenteric countries; (ii.) the tongue is generally characteristic, being very clean, red, and often sore; (iii.) the stools vary, but are generally pale, pasty, frothy, and easily ferment; (iv.) slight errors in diet produce great aggravation of the diarrhœa. The *treatment* consists almost entirely in regulating the diet. Only milk, farinaceous food, and eggs should be allowed; no meat, vegetables, or fruit. In severe cases the patient should live entirely on boiled skimmed milk, 4 or more pints a day. Rest and warmth are very important. Bismuth, with or without a little opium, may check the diarrhœa.

VI. **Nervous Diarrhœa** is a form of diarrhœa which may

<sup>1</sup> Golding Bird, Report of Clin. Soc. Lond., *The Lancet*, June 1, 1902.

continue for years; it occurs in nervous people, and has the following characteristics: (i.) the motions are often quite healthy, sometimes liquid, never attended by melæna or mucus. There is usually no pain or tenesmus. The diarrhœa is generally recurring, or intermittent, occurring in the early morning, or when the patient is "nervous." Sometimes it follows each meal (*lienteric diarrhœa*). (ii.) It occurs, for the most part, in females of a neurotic type. (iii.) Diet seems to produce little or no influence, but *the attacks are determined* by mental emotion or bodily fatigue. A plain but generous diet is called for; and the administration of nux vomica, belladonna, and bromides are often more efficacious than astringents. Careful search should be made for any source of uterine or other reflex irritation. Arsenic (M ij. Fowler's solution), with meals, is said to be a specific for *lienteric diarrhœa*.

The crises of LOCOMOTOR ATAXY sometimes take the form of acute diarrhœa, with or without pain. In HYSTERIA, acute attacks of diarrhœa with noisy borborygmi may occur—determined in the same way as other hysterical attacks.

VII. **Amyloid disease** of the intestines gives rise to a most intractable form of chronic diarrhœa. Indeed this is the common mode of death in amyloid disease of the viscera. The characteristics here are: (i.) a history of long-standing purulent discharge, or of syphilis; (ii.) great pallor of the skin, accompanied by evidences of lardaceous disease in the spleen, liver, and kidney; (iii.) the stools are generally liquid and extremely offensive, sometimes attended by hæmorrhage. The *treatment* is very unsatisfactory. Pernitrate of iron, sulphuric acid, logwood, acetate of lead may be tried, and also opium, which does no harm even when there is amyloid disease of the kidney, as there is no tendency to uræmia.

The *rarer* causes of chronic diarrhœa are:—

VIII. **Senile Diarrhœa** was, I believe, first described by MacLachlan, in his "Diseases of Old People." It occurs in persons over 60 or 70, and is very chronic in its course, but the patient suffers very little. Careful examination for organic disease should be made before concluding that the condition is simply senile diarrhœa. Astringents and most other remedies fail to relieve it, and it may exist for many years without emaciation or danger to life.

IX. **Chronic catarrhal enteritis** is an affection frequently overlooked. The patients suffer at intervals from (i.) attacks of diarrhœa alternating with constipation; (ii.) mucus is found in the stools during the periods of diarrhœa; (iii.) the general health during the attack is

lowered, with despondency, and other symptoms of nervous prostration, together with (iv.) great abdominal discomfort and a sense of sinking. These attacks last only a few days, and occur after slight irregularities in diet, such as the eating of game, or after a long railway journey, or drinking a water different from that to which the patient is accustomed. Sometimes the history dates from a severe attack of acute diarrhœa. Relapses may continue for 10 to 12 years or longer. These cases have to be diagnosed from portal obstruction and from ulceration of the bowel. The *treatment* consists in avoiding those things known to produce the attacks, and in administering during the attack first a small dose of castor oil, then small doses of astringents, bismuth, and alkaline carbonates.

X. **Mineral poisons**, and especially arsenic and antimony, in small continuous doses may cause persistent diarrhœa. It was in this way that the celebrated Maybrick case was discovered.

XI. **Pancreatic disease** has been associated with diarrhœa. Dr. Burney Yeo has described a case of chronic diarrhœa which resisted all treatment until pancreatic emulsion was administered. The diarrhœa returned when this was stopped, and ceased again on its administration. It may be assumed that only the chronic forms of pancreatic disease (*e.g.*, Fibroid Pancreatitis) would be attended by this symptom, and the diarrhœa is probably dependent upon the excess of fat and undigested muscle fibre in the fæces (§ 182).

§ 224. (XII.) **Psilosis** or **Sprue** is a condition met with in the tropics. It is characterised by diarrhœa and other symptoms of congestion of the alimentary canal, usually running a prolonged, and often fatal, course.

The *Symptoms* consist of (i.) diarrhœa, which is very chronic and continuous, and attended by pale, copious, and frothy stools; (ii.) there are also symptoms of dyspepsia, with distension of the abdomen; and (iii.) tenderness of the mouth. At times these symptoms are exacerbated, and aphthous patches appear on the mucous membrane of the mouth and pharynx. In the course of time the patient becomes extremely feeble. Europeans in the tropics are often affected with slight diarrhœa, and Sprue is apt to supervene upon this. Any disease of the intestine, such as dysentery, and any cause of general weakness predisposes to Sprue. Women are more often affected than men.

The *Diagnosis* is made by the history of chronic diarrhœa, with pale copious stools, and the tender state of the mouth. If untreated the disease is usually fatal in one or two years. Even with treatment it may lead to death in 6 to 10 years. Much depends upon the age of the patient; if in late middle-age cure is unlikely.

The *Treatment* consists in giving as much rest as possible to the alimentary canal. The patient must be put to bed and kept on a restricted milk diet for six weeks or more. No solid food must be allowed. The patient may gradually return in the course of a few months to ordinary diet, but meat and coarse vegetables must be taken only seldom, even after recovery. When milk disagrees, after trying condensed and peptonised milk, or milk and lime water, raw meat juice may be tried for a time, and then gradually return to milk. It is important not to give large quantities per diem, however much the patient may complain of hunger. To begin with only 2 pints of milk should be allowed; this may be increased, when the mouth is not tender, to 5 or 6 pints a day, in the course of two months.

§ 225. **Tenesmus** literally means straining at stool (τείνω, to strain or stretch); but in its widest sense it may be taken to mean any local rectal sensation of "bearing down" which results either in a constant desire to go to stool, or a straining when at stool. The latter may lead to prolapse of the rectum, especially in children. Diarrhœa is always attended by more or less tenesmus, but tenesmus is not always attended by diarrhœa. (1) Ascertain if the tenesmus is accompanied by diarrhœa—*i.e.*, are



the motions frequent and liquid. If so, refer to the section on Diarrhœa, § 219. (2) Particular attention should also be paid to the shape and consistence of the motions. (3) Examine locally for any anal or rectal condition such as fissures, piles, polypi, or ulcers. All the pelvic organs should also be very thoroughly investigated, especially in women, in whom the symptom is commoner than in men.

*Causes.* Tenesmus (without diarrhœa) may arise from four groups of causes :—

1. Various conditions of the ANUS—pruritus, eczema, or fissure, may be overlooked for a long time, as in the case mentioned in § 223, II. Piles also, if internal, may be difficult to detect, but streaks of bright blood will appear in the motions from time to time in that case.

2. Various RECTAL CONDITIONS, especially stricture or ulceration. The former (usually of syphilitic origin) is attended by tape-like stools ; the latter is attended by pus or blood or both. Prolonged use of purgatives, or the constant use of the glycerine enema may result in straining at stool and prolapse of the rectum. Proctitis (inflammation of the rectum and anus) is another cause. In the aged, we should always suspect cancer of the rectum, although this is usually attended by actual diarrhœa.

3. PRESSURE ON, or irritation of, THE RECTUM FROM WITHOUT such as may be caused by chronic congestion, version, or other disease of the uterus. These in women, and congestion or new growth of the prostate in men, are both very common causes. Any bladder disease such as stone—a frequent cause of tenesmus in children, and apt to result in prolapse of the rectum—or new growths. Chronic cystitis may cause this distressing condition. Ischio-rectal abscess, pelvic hæmatocele, and various ovarian and Fallopian tube lesions are all apt to cause tenesmus. The catamenial period is almost invariably attended by a certain amount of tenesmus ; and in many women the bowels are only properly opened at such times.

4. In HYSTERICAL AND NERVOUS SUBJECTS any fright or other emotion may at once determine tenesmus, which is spoken of by the patient as “diarrhœa.” In *tabes dorsalis* the “rectal crises” may take the form of tenesmus.

*Treatment.* The indications consist of—(1) the removal of the cause; the treatment of piles and other causal conditions being found elsewhere. (2) The relief of any local congestion or irritation of the rectum. Fissure may require antisyphilitic remedies. In any case morphia, belladonna, hydrochlorate of cocaine in the form either of suppositories or enemata will relieve the distress from which the patient suffers (F. 73 is useful).

§ 226. **Blood in the stools** is met with, as we have seen, in dysentery and some cases of simple diarrhœa; but it may be met with unassociated with the latter. The presence of blood in the stools may be recognised by the reddening of the water in which the stool is placed; or by the spectroscope. Clinically, blood in the stools may present two widely different characters: (a) When the blood is of *bright crimson colour* it indicates either that the bleeding comes from the rectum or the lower part of the large bowel; or, if it comes from the upper part of the intestinal canal, that it is too large in amount to be acted upon by the intestinal secretion. (b) *Melæna (tar-coloured stools)* is met with when hæmorrhage in moderate quantity has taken place in the stomach or the upper part of the alimentary tract, when the digestive fluids of the stomach and intestine acting on the blood give it this tarry colour. The causes of these two conditions may to some extent be interchangeable, for what will produce a large hæmorrhage at one time may at another produce only a little.

(a) **Bright red blood** may be due to the lesions of the lower part of the alimentary canal. Of these, 1, 2, 3, 5, and 7 are referable to the anus or rectum, and may generally be discovered on local examination.

1. **HÆMORRHOIDS**, or Piles, are undoubtedly the commonest cause of blood in the stools. The blood is generally met with in streaks only; but the quantity may at other times be very large. This condition is fully described below, § 227.

2. **FISSURE OF THE ANUS** may also produce streaks of blood. It is a not infrequent condition, and is recognised by the excruciating pain during and after defæcation. The irritation it causes may give rise to a variety of false diarrhœa, as in the case narrated in § 223. The fissure can always be seen by *careful* examination.

3. RECTAL ULCERS may give rise to streaks of bright blood in greater or less quantity, mixed with pus and mucus. They are usually of syphilitic, cancerous, or dysenteric origin, and can frequently be felt by digital examination (§ 223).

4. A discharge of blood-stained mucus, coming on somewhat suddenly in a child, is highly suggestive of *INTESTINAL INTUSSUSCEPTION*, which is one of the causes of acute obstruction (§ 230).

5. RECTAL POLYPI are also met with chiefly in children.

6. *TYPHOID* and *TUBERCULAR ULCERATION* of the small intestine sometimes produce very profuse discharges of bright red blood which comes from the lower end of the small intestine. Other evidences of these affections are present.

7. *BILHARZIA HÆMATOBIA* causes hæmaturia, but it also gives rise to fleshy masses in the rectal mucous membrane, resembling piles which bleed. The ova are very characteristic, and can be detected in the fæces (§§ 217 and 301). It is only met with in persons who have been to South Africa or other tropical or sub-tropical countries.

8. *VARIOUS GENERAL BLOOD CONDITIONS* may give rise to hæmorrhage coming from the rectum or elsewhere in the alimentary canal in varying amount. This is so in purpura, scurvy, hæmorrhagic forms of the specific fevers, acute yellow atrophy of the liver, leukæmia, etc.

(b) *Melæna* (*tarry stools*) is met with when bleeding takes place from the stomach or high up in the alimentary tract in moderate quantity. Its causes are as follows :—

1. When coming FROM THE STOMACH, it may be associated with profuse *hæmatemesis* (§ 193); the commonest causes of hæmatemesis are gastric ulcer and hepatic cirrhosis.

2. *PORTAL OBSTRUCTION* (§ 235) is one of the most frequent causes of melæna, especially that form due to alcoholic cirrhosis of the atrophic variety. It may also occur with the engorged liver and spleen of advanced cardiac disease. In either case the hæmorrhage in these circumstances is a natural safety-valve, and gives relief to the engorged state of the portal circulation.

3. *CANCEROUS, TUBERCULAR, and other ULCERATIONS* of the small intestine (see § 223) and lardaceous disease of the bowel may also produce melæna.

4. The GENERAL BLOOD CONDITIONS above named, when the hæmorrhage is small in amount, are attended by tarry instead of bright red stools. A very rare condition, apparently due to some blood change, in *infancy*<sup>1</sup> is attended by melæna and some pyrexia. It starts the first week of life, and terminates fatally in a few days. Nothing has been found *post-mortem*.

5. The ANKYLOSTOMUM DUODENALE (ankylostomiasis) is a frequent cause of profuse melæna, so profuse and continuous as to cause anemia, in Egypt and other foreign countries (§ 217).<sup>2</sup>

The *treatment* of melæna should be directed to the cause, but the general principles are those laid down for hæmâtemesis (§ 193). Turpentine (℥ x. capsule), lead acetate, and opium are recommended. Supra-renal gland has recently been advocated as a remedy.<sup>3</sup> *Ankylostomum* is readily destroyed by thymol. First empty the bowels by a brisk purge given the night before, then administer 3 or 4 doses of thymol, 20 grains each in cachets, at intervals of 3 hours. The bowels must be kept freely open all the while, and alcohol carefully avoided so as to prevent absorption and poisonous effects.

§ 227. **Hæmorrhoids**, or Piles, consist of a varicose condition of the rectal veins. This varicosity forms a swelling of variable size, which may be altogether within the anus (internal piles), or partly internal and partly external. Internal piles may in some cases be seen, when the patient "bears down," as small purple swellings just protruding from the sphincter; in other cases internal piles are discovered only on digital examination of the rectum.

*Symptoms.* (1) Streaks of *bright red* blood occur in the stools, and sometimes as much as  $\frac{1}{4}$  pint of blood may be passed at one time. (2) There is pain on defæcation, the pain continuing for some time after the passage of a stool. When a pile becomes inflamed, or strangulated by the sphincter, severe pain and discomfort is experienced, and the patient may have to remain in bed for days. Pain may be referred to other parts of the body—*e.g.*, to the testicles, bladder, or loins. (3) Constipation nearly always accompanies piles, due partly to mechanical obstruction, and partly to the pain caused by defæcation. (4) In severe cases

<sup>1</sup> Osler, "Prin. and Pract. of Med.," p. 346, 2nd edit.

<sup>2</sup> Other worms are mentioned by Dr. F. M. Sandwith in *The Lancet*, vol. ii., 1899.

<sup>3</sup> *Brit. Med. Journ.*, Nov. 3, 1900.



constitutional symptoms are developed, such as lassitude, irritability, headache, faintness, and later on, perhaps, anæmia, from loss of blood.

*Etiology.* (1) Portal obstruction is itself a cause of piles, and in all marked cases we should seek for the other symptoms of this lesion (§ 235). (2) Habitual constipation, however, is undoubtedly the most common cause of hæmorrhoids, particularly in women, who in early life are so apt to contract this habit. (3) Alcohol, especially in the form of malt liquors, with excess of sugar, cause portal congestion, and thus become a source of piles. Alcohol in any form aggravates the condition. (4) Sedentary occupations, and deficient exercise, also produce piles. (5) Various local conditions, such as sitting on soft cushions, which constrict the inferior hæmorrhoidal veins, uterine displacements, pelvic and other tumours, are all potent causes of hæmorrhoids.

*Prognosis.* Hæmorrhoids are not usually regarded as serious, but they may be extremely troublesome, partly by the constant loss of blood, partly by their liability to repeated attacks of inflammation, and partly by the pain they cause.

*Treatment.* Much may be done by three simple means : (1) The avoidance of alcohol (especially malt liquors), (2) keeping the piles scrupulously clean, and (3) the bowels regularly and loosely open. Rich food and other causes of hepatic congestion must be forbidden. Hydragogue purgatives are best—such as Hunyadi, Friedrichshall, or Carlsbad waters every morning, or confect. sulph., or sennæ, with an occasional cholagogue at night. Regular exercise is desirable. Local applications should be of the simplest kind. The old-fashioned gall and opium ointment is now very properly replaced by hamamelis, with conium, morphia or cocaine for the pain if necessary. Ung. hamamelidis (B.P.) is an excellent preparation, and is best applied on a strip of lint inserted within the anus and left there; or a suppository may be employed, containing 1 to 3 grains of hamamelin, and  $\frac{1}{8}$ th grain of morphia if requisite. Inflamed piles are very painful, and are best treated by warm hip-baths, frequent bathing, sitting over hot water in a bidet, warm fomentations with opium, belladonna or cocaine. Incision may be required, but leeches are better. For the radical cure removal by surgical measures is called for in some cases.

§ 228. **Intestinal worms** may give rise to no symptoms at all. They are most frequently met with in children; and may remain undiscovered until they are found in the stools. The morphology, symptoms, and habitat of the various entozoa are described in Table XIX., p. 386. Thread worms (Fig. 68) and round worms (Fig. 69) are the most common. It used to be considered that thread worms lived in the colon, but it is now believed they exist in the caput cæcum, and sometimes in the vermiform appendix. This fact explains those cases which appear to be cured for a time, but which continually relapse.

The *symptoms* are very indefinite, and consist of: (1) vague and persistent, though often paroxysmal pains in the abdomen; (2) capricious and sometimes ravenous appetite, in spite of which the child becomes thin and sallow; (3) grinding of the teeth at night, picking of the nose, and other reflex phenomena; (4) irregularity of the bowels, or diarrhœa.

The *treatment* differs for the different worms. For *thread worms*, the best treatment consists of quassia injections. After an aperient, one ounce of powdered quassia to a pint of boiling water is when cold injected slowly into the bowel and retained as long as possible. Common salt injections of the same strength may be used. The worms are destroyed with two or three such injections. Where the worm has its habitat high up in the intestine (as the *tape-worm*), treatment is conducted in three stages. (1) In order to starve the parasite by keeping the alimentary canal as empty as possible, the patient should have no food after midday, and at night or the next morning a purgative must be taken. This leaves the worm uncovered, and thus readily acted upon by (2) the anthelmintic, which is given about an hour after purgation. The chief anthelmintic is ext. filix mas liq. one drachm. Some recommend ℞ xxx. sp. turpentine to be given with this; others give kousso (4 dr.) or pelletierine (2 gr. of the alkaloid). (3) Two hours later give calomel with a saline aperient, to eject the worm from the body. The stools must be examined to see that the head is passed. If only segments are passed, the worm will grow again, and the same treatment will have to be repeated within three months. For the *round worm* the specific remedy is santonin, given in two grain doses to a child of three and upwards; for an adult five grains are given. For *ankylostomum duodenale* see p. 408.

§ 229. **Constipation** is insufficient action of the bowels, or the passage only of hard, dry, or ball-like masses of fæces (scybala), independent of organic disease within or outside the intestinal canal. This source of fallacy must be carefully excluded before diagnosing a case as one of simple constipation.

The *symptoms* which accompany or result from constipation are sufficiently familiar—at first headache, languor, and depression, followed by a furred, coated tongue, dyspepsia, anæmia, sleeplessness, and eruptions, for the most part of an urticarial or erythematous nature. The temperature may rise a degree or so in certain conditions from temporary constipation, and I have met with one case where it went up to 102°. The retention of hard faecal masses

may give rise to an alternating diarrhœa, which leads to error in diagnosis. Hemorrhoids is another consequence of habitual constipation, and a distended ulcerated colon may result in some subjects. In women, in whom the condition is far more common than in men, a chronic torpidity of the bowels may predispose or even lead to uterine disease; and in both sexes varicose veins, œdema of the legs, sciatica, especially on the left side, and numbness of the legs are among its consequences.

For purposes of treatment we may consider the **causes** of simple or uncomplicated cases of constipation under three headings—

a. **Errors of diet.**

- i. Too bland food—*e.g.*, no vegetables, no food with coarse residue.
- ii. Too dry food—*e.g.*, deficient fluid ingesta.
- iii. Too little or poor food, or too great uniformity of diet.

b. Causes of **defective vermicular action** (other than errors of diet).

- i. Sedentary habits.
- ii. Advanced age, and other conditions where the general neuromuscular tone is poor, as in melancholia, anæmia.
- iii. Prolonged disregard of calls of nature.
- iv. Weak abdominal muscles.
- v. Atony of the intestine, with or without chronic (mucous) colitis.
- vi. Diseases of the cord or brain—*e.g.*, cerebral tumour, tabes.
- vii. Some febrile states.
- viii. Spasmodic reflex conditions, as from uterine or ovarian diseases.
- ix. Drugs, such as opium, lead, iron.

c. **Deficiency of bile or intestinal secretions.**

- i. Functional inactivity of the liver, § 251.
- ii. Profuse vomiting.
- iii. Excessive loss of fluid by skin or kidneys.
- iv. Astringents, such as chalk or catechu. Hard waters also act in the same way.

*Treatment.* Chronic constipation is only serious in respect of the troublesome consequences mentioned above. In its treatment we should first endeavour to *find out the cause*. Examine the colon to see if it be distended; place one hand at the back and press it forwards between the iliac crest and the last rib to meet the other hand, which is placed flat on the anterior abdominal wall, the patient being in the recumbent position. Having excluded local causes by a thorough examination, we should consider the various causes above mentioned. The treatment of constipation comes under six headings.

- (1) *Dietetic treatment*—increase the amount of fluid taken, *e.g.*,

by sipping a tumbler of hot water slowly whilst dressing in the morning and undressing at night. Avoid large quantities of milk, or hard water. Coarse foods should be eaten which stimulate the intestinal wall, such as oatmeal, wholemeal or brown bread, green and raw vegetables, figs and ripe fruits. A teaspoonful or tablespoonful of salad oil at meal-times will often be very efficacious in cases due to deficient intestinal secretion. (2) Inculcate regular *habits*, even when there is no inclination to go to stool. (3) Active *exercise* is advisable excepting where uterine or ovarian irritation is in operation. A modified form of exercise may be practised by lying flat upon the back and rising from a recumbent position without the aid of the arms ten or a dozen times each morning. (4) Abdominal *massage* is often useful. Gently "rolling" the abdominal wall, or rolling a 7-lb. ball over the abdomen, in the direction of the hands of the clock.<sup>1</sup> (5) *Drugs*—for occasional constipation, aloes with the evening meal and a seidlitz powder in the morning are the most harmless. Calomel or other mercurial preparations should not be given habitually, but may be taken once a week for a few weeks. A useful vegetable pill is pil. col. co., pil. rhei co., aa gr. j., ext. hyoseyami, gr.  $\frac{1}{2}$ ; two at bedtime. Belladonna and nux vomica in small doses undoubtedly promote vermicular action—the former is especially useful for women with pelvic irritation. A two months' course of cascara sagrada, graduating the dose to the individual, will often break through a vicious habit of constipation. Jalap, elaterium, scammony, and gamboge are useful where drastic purgation is desired. Salines given daily for some weeks will often re-establish the functions of a torpid intestine (F. 46). These may be given in the form of the mineral waters, such as Carlsbad, which contains 13 grains of sulphate of soda to the tumbler with alkalis (dose, one or two tumblers twice daily); Friedrichshall, which contains 60 grains of the sulphates of magnesia and soda with alkalis (dose, half a tumbler full daily); Hunyadi water, which contains 200 grains of sulphates of soda and magnesia with alkalis (dose,  $\frac{1}{4}$  to  $\frac{1}{2}$  a tumbler). All of these are best given on an empty stomach (F. 57, 88, and 90 are also useful). An excellent aperient for children is cascara and maltine mixed together in the proportion

<sup>1</sup> Herschel. "Constipation." London. 1899.



of 10 to 20 ℥ of the ext. casc. liq. to the teaspoonful of maltine. (6) *Enemata* are useful in certain conditions, though it must always be borne in mind that they do not empty the small intestine. They are, however, better than constant purgation in some cases. The ordinary soap enema or one or two pints of soapy water may be used. Half an ounce of glycerine is a very effective enema, but it should not be used longer than a few weeks, for it produces an irritable condition of the rectum. In cases of very prolonged constipation which resist all other means, I am in the habit of prescribing a quarter to half a pint of olive oil as an enema every night. If this be injected very slowly it is retained, and after a course of one or two weeks it is wonderful how regularly the bowel resumes its functions. Another valuable enema consists of the following: mix two tablespoonfuls of sod. bicarb. with two tablespoonfuls of olive oil, adding just sufficient warm water to make a cream. Then stir in one pint of hot water, and inject slowly into the rectum, with the patient on his back.

*The patient complains of* SUDDEN STOPPAGE OF THE BOWELS *with inability to pass even flatus, ABDOMINAL PAIN, and VOMITING which gradually becomes stercoraceous; his PULSE IS RAPID and there is a tendency to COLLAPSE*—the case is probably one of ACUTE INTESTINAL OBSTRUCTION.

§ 230. **Acute intestinal obstruction** is one of the most serious medical or surgical emergencies to which a medical man can be summoned.

The *symptoms common to all forms* of acute obstruction are—(1) *complete constipation, not even flatus being passed.* (2) The *pain* is at first paroxysmal, referred to the umbilicus, though it becomes continuous later on. There is not usually much tenderness. (3) The *vomiting* comes on earlier and is more urgent, and becomes more rapidly stercoraceous in proportion as the obstruction has taken place high up in the intestines. (4) Abdominal distension is generally present, and this may be one-sided, so giving us a clue to the position of the obstruction. (5) Constitutional symptoms gradually supervene, with prostration and a thready, *rapid pulse.* These also are more urgent when the small intestine is involved. The urine is diminished in proportion

as the obstruction is near the stomach, for then the vomiting is more urgent.

*Diagnosis of acute intestinal obstruction.* When summoned to a case presenting these three symptoms—stoppage of the bowels acute abdominal pain and vomiting, the first step is to identify the case as one of acute obstruction. In *colic* (renal, hepatic, or intestinal), all of these three symptoms may be present, but the patient's general condition is not so serious, and the bowels are readily relieved by purgatives or enemata. Moreover, the position of the pain in renal and hepatic colic is characteristic (see § 173). In *acute peritonitis* there is great tenderness over the abdomen, thoracic respiration and some fever (see also § 171). But when there is *perforation* into the *peritoneum*, collapse is present, at first without fever; and perforation is diagnosed with difficulty only by (i.) the passage of wind by the bowel; (ii.) the collapse being much greater than that even in acute obstruction; and (iii.) a possible history of the condition which has resulted in perforation or rupture (consult also § 170). It is sometimes impossible to diagnose these two conditions, and an exploratory operation should be undertaken without delay.

*Causes of intestinal obstruction.* It is of some importance to ascertain the cause, for the prognosis and treatment differ somewhat in each case. (a) In *acute* intestinal obstruction, in which the symptoms come on *suddenly* in a person previously healthy, there are four *common* causes—(I.) external hernia, (II.) intussusception, (III.) internal strangulation, (IV.) appendicitis (perityphlitis). (b) Sometimes, however, acute will supervene on chronic obstruction, and the *common causes of chronic obstruction* (§ 231) are also 4 in number—(I.) malignant stricture of the bowel, (II.) simple stricture, (III.) pressure of a tumour, and (IV.) dilatation of the bowel.

*Features special* to the several causes of acute intestinal obstruction.

I. EXTERNAL HERNIA is known by the presence of a tumour in the femoral, inguinal or umbilical region. No impulse on coughing is present. Obturator hernia is very rare and can only be discovered at the time of operation.

II. INTUSSUSCEPTION, or invagination of the bowel, is by far the

commonest cause in childhood. According to Brunton, it is a cause of 43 per cent. of all cases of obstruction. True intussusception is always from the bowel above into the part below, and in more than half of the cases the lower part of the ileum becomes invaginated into the cæcum. In a third of the cases some other part of the ileum, and in about one-eighth some part of the colon, is implicated. The invaginated portion slowly sloughs, the two edges may be welded together, the slough may pass about the eighth or tenth day, and thus spontaneous recovery may occur, though this is relatively rare. Death from perforation and collapse is more usual unless the case is dealt with surgically. Intussusception is known by (i.) severe tenesmus; (ii.) a rectal discharge of *blood and mucus*; (iii.) a sausage-shaped tumour may be felt, altering in position, on palpating the abdomen, and in extreme cases the invaginated portion of bowel is felt per rectum; and (iv.) the patient is a child.

III. INTERNAL HERNIA OR STRANGULATION, *e.g.*, by bands of adhesion,<sup>1</sup> is known by (i.) the urgency of the symptoms, (ii.) the patient is an adult man, with (iii.) a history of old peritonitis. VOLVULUS (or twisting of the bowel) may be indistinguishable from the preceding—indeed it practically results in strangulation—but (i.) it occurs in men over 40, usually with a history of chronic constipation, (ii.) abdominal distension may be great, (iii.) sometimes a tumour is felt over the sigmoid flexure, the usual site of volvulus.

IV. APPENDICITIS (Perityphlitis) is known by (i.) pain and tenderness of a constant character in the right iliac fossa, (ii.) the temperature is elevated if the inflammation has extended to the parts around, (iii.) a history of previous attacks is often present (see § 176).

The rarer causes of acute obstruction are three in number:—

V. IMPACTION IN THE BOWEL of a large GALL-STONE. This is not so rare as might be supposed, and Dr. Murchison was able, without much difficulty, he stated, to collect 34 cases.<sup>2</sup> A large gall-stone escapes from the gall-bladder by ulceration into the bowel. The obstruction is

<sup>1</sup> Internal strangulation may also arise from: (1) Adhesion of the end of the appendix vermiformis through which a knuckle of the bowel gets nipped. (2) Agglutinations of the bowel. This is a cause of  $2\frac{1}{2}$  per cent. of the cases of acute obstruction. (3) Congenital deficiencies in the mesentery or bowel, or the foramen of Winslow. (4) The rarer conditions are connected with congenital malformations. For instance, an interesting case of patent Meckel's diverticulum into which the posterior wall of the ileum became intussuscepted forming an umbilical tumour in a male child at six weeks, is published by Dr. Leonard Guthrie in "Pediatrics," vol. ii., July 1, 1896.

<sup>2</sup> Lectures on Diseases of the Liver, by Charles Murchison, M.D., LL.D., F.R.S., 2nd ed., Lond., 1877, p. 497.

high up in the small intestine, and consequently (1) The pain and constitutional symptoms are of extreme severity, and of very sudden onset. (2) The patient is usually a female (four females to one male), at or beyond middle age. (3) There may be a history of biliary colic, and in all cases there is a history of localised peritonitis some weeks or months before the seizure. (4) The symptoms may intermit. from the stone shifting its position.

VI. Obstruction of the bowel may sometimes be due to an EXTRAVASATION OF BLOOD into the coats of the intestine. It occurs only in purpura, hæmophilia, and other blood disorders. Such cases are recognised by evidences of hæmorrhage in other positions—melæna, epistaxis, purpura.

VII. Among the still rarer causes of obstruction may be mentioned masses of round worms (Trousseau), concretions of ammonio-phosphate of magnesium (a frequent cause in horses though rare in man), and other foreign bodies in the intestine.

*Clinical investigation and diagnosis of the cause of obstruction.*

If the case occur in a child, and there is a history of diarrhœa for the past few days, it is almost certainly intussusception; in an old person suspect rectal stricture, impacted fæces, or volvulus; in a young adult suspect strangulation, hernia, or perityphilitis. If the vomiting come on early and is urgent it points to a tight constriction *high up* in the intestinal tube. If the distension is chiefly in the centre of the abdomen, the obstruction is probably above the ileocaecal valve; if it is chiefly in the flanks, the obstruction is below the valve; if more in the right than in the left flank, the obstruction is probably in the sigmoid flexure.

When called to such a case first examine for swelling in the positions of external herniæ. If the abdomen be distended, and presents visible waves of peristalsis, inquire as to the causes of chronic obstruction (*infra*), as the case is probably an acute supervening upon a chronic obstruction. Always *examine per rectum*, for in acute intussusception the invaginated part of the bowel may be felt per rectum, and there may be a discharge of blood and mucus; or a stricture or other cause of chronic obstruction may thus be discovered. Next inquire into the past history, *e.g.*, for peritonitis (as this is a cause of internal strangulation), or for appendicitis or hepatic colic. Then examine the abdomen by palpation and percussion for tumour or tenderness. If the abdomen is distended only on one side the site of the obstruction may be localised.

*Prognosis.* The prognosis of obstruction of the bowels is always very serious. Death occurs in the natural course either from



(1) gangrene and rupture of the bowel, or (2) exhaustion and collapse. The prognosis almost entirely depends in the present day upon the *stage at which the case comes under notice*, and the treatment adopted. All the acute cases will probably necessitate early surgical interference and a surgeon should be summoned at once, so as to be prepared at any moment to explore the abdomen if the obstruction be unrelieved. The success and justification of such diagnostic operations form one of the chief triumphs of modern surgery. As regards the *causes*, obstruction from a gall-stone is perhaps the most serious, then intussusception, then internal strangulation; perityphlitis being most favourable. Among the gradual causes, carcinoma of the bowel gives the gravest prognosis, and paralysis the most favourable. Cases in which the obstruction is high up are less favourable than those in the large bowel.

*Treatment.* Acute intestinal obstruction is one of those serious conditions that demand the resources of both a physician and a surgeon, who should jointly undertake the management of a case. The indications are:—(1) to ascertain the cause; (2) to endeavour to remove the obstruction; and (3) in the meantime to support the strength and relieve the pain by controlling the peristalsis upon which it depends. Enemata may be given in all cases; purgatives should be avoided. Warmth is applied to the abdomen in the form of hot fomentations, turpentine, belladonna or opium stupes. If there are signs of peritonitis cold is said to be more efficacious. The question of the administration of opium is debated (see appendicitis). Surgeons object that it masks the symptoms in cases likely to require operation, yet it is hard to know how otherwise to relieve the extreme pain in these cases. The diet should consist of fluids, such as iced milk, beef tea, and stimulants, given in small quantities, and frequently.

In *external hernia*, after a warm bath, antispasmodics, and taxis under chloroform, proceed at once to operation. In *intussusception*, some mild cases have a tendency to spontaneous recovery. The patient should be inverted, and large injections given per rectum. Thus, olive oil injections ( $\frac{1}{2}$  a pint) may be given very slowly, followed half an hour later by the slow injection of 1 or 2

pints of gruel. The same effect, the forcing back of the intussusception, may be produced by copious warm water injections or blowing up the bowel with bellows under chloroform. These methods failing, and the symptoms becoming graver, laparotomy is indicated; most surgeons advocate this procedure at latest 24 to 36 hours after the onset of the condition. In *internal strangulation* or twisting, it is best to operate without delay if an injection does not relieve and we are certain of the diagnosis. In cases of recovery without operation, there has probably been a simple volvulus. But death almost always occurs in cases of internal strangulation if unrelieved. Manipulation, and inflating the bowel by means of bellows, have been suggested, but there is considerable risk attending these procedures. In *appendicitis*, see § 176. In *impacted gall-stone*, the progress is so rapid towards a fatal issue that operation, if undertaken, must be done immediately. The same remark applies to other foreign substances in the intestine.

*The patient complains of CONSTIPATION progressively increasing until he is unable to pass even flatus, ABDOMINAL PAIN, and from time to time VOMITING, there is a rapid pulse, and general ill-health—the case is one of CHRONIC INTESTINAL OBSTRUCTION.*

§ 231. In **Chronic intestinal obstruction**, (1) the abdominal pain is generalised, intermittent, and of increasing severity. (2) There is constipation, or a history of alternate constipation and diarrhœa culminating in complete stoppage; and (3) abdominal distension in most cases, and peristalsis in some, may be visible. The chief causes of this condition are 4 in number—

I. **MALIGNANT STRICTURE** by new growth in the wall of the bowel—*e.g.*, cancer. Its most common situations are the colon, especially the sigmoid flexure, and the rectum. This cause of obstruction may be recognised by (1) the presence of a tumour or stricture which may be felt on examination per rectum, and the distension of the abdomen being most marked in the flanks. When the tumour is situated higher up than the sigmoid flexure it may generally be felt through the abdominal wall. (2) When the sigmoid flexure or rectum is affected, the illness is often preceded by sciatica on the l. side. (3) There are cancerous

cachexia, the age of the patient, and perhaps hæmorrhage and fœtid discharge to aid in the diagnosis.

II. SIMPLE, *i.e.*, NON-MALIGNANT STRICTURE of the intestine may arise in consequence of dysenteric, syphilitic, or other ulceration, either in the colon or in the rectum. An ulcer alone is capable of producing symptoms of obstruction. This cause is recognised by—(1) the absence of a tumour, and (2) a previous history of dysentery (perhaps only a mild attack), and residence in a tropical climate; or a history of syphilis, with a rectal discharge.

III. PRESSURE ON THE BOWEL by a TUMOUR or an enlargement of some viscus such as the uterus. This cause is recognised by the physical signs of tumour or enlargement respectively.

IV. DILATATION OF THE BOWEL from paralysis of its coats. This is chiefly met with in the aged. It is differentiated from the other causes chiefly by (1) the absence of cachexia, tumour, emaciation, or other symptoms of the preceding causes, and an absence of a history of syphilis or dysentery. (2) The gradual formation of a soft fecal tumour, situated in the descending colon. The *diagnosis* of these causes is also discussed on p. 416.

V. CHRONIC PERITONITIS (§ 177) causes a matting together of the intestines, and intestinal obstruction may result. Cancerous peritonitis is attended by much pain and the effusion of much fluid; but in tubercular peritonitis there are mostly adhesions, less pain, and less fluid.

VI. CHRONIC INTUSSUSCEPTION is known by—(1) it occurs usually in children; (2) tenesmus is present; (3) a tumour may be felt with characters similar to that met with in acute intussusception (*q.r.*); and (4) there is usually no marked distension: see also acute intussusception (above).

VII. APPENDICITIS (§ 176) may occasionally cause chronic, but more often acute, obstruction.

*Prognosis.* In all forms of chronic intestinal obstruction the symptoms of acute obstruction are apt at any time to supervene, from impaction of feces above the narrowing lumen of the gut, but apart from this the prospect differs considerably in the different causes. A cancerous stricture is the most, a dilated colon the least serious. Syphilitic stricture may be relieved by iodides, dysenteric stricture is much graver and irremediable. The course of a tumour varies with its nature. Chronic intussusception *may* spontaneously resolve, the invaginated part sloughing off and being passed by the rectum, but the outlook is always grave.

*Treatment.* In most of the cases of chronic intestinal obstruction,

surgical procedure is ultimately necessary, but at first the treatment consists in watching the patient until a diagnosis can be formed with as much accuracy as possible, and in giving digestible food, preferably such as leaves but little residue; the pain being relieved by opium and external applications (hot fomentations with turpentine or opium). In atony of the bowel, if oil enemata and other medicinal treatment fail, the fæces may require to be removed by mechanical means (scooped out). For simple stricture of the rectum gradual dilatation by bougies may be tried. In chronic intussusception keep the patient under observation, giving enemata and digestible foods. In cancerous stricture an operation may prolong life by the formation of an artificial anus, and the longer the operation is delayed, the worse is the prognosis for recovery. It should never be delayed until vomiting has commenced. In some cases the bowel has been resected with success. The injection of mixed toxines, which have lately come into vogue, is worth bearing in mind in this connection.<sup>1</sup>

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<sup>1</sup> "The Treatment of Sarcoma and Carcinoma by Injections of Mixed Toxines," by C. Mansell Moullin, M.D. Oxon., F.R.C.S. London, 1899.



## CHAPTER XII.

### THE LIVER.

WE still remain in comparative ignorance of the functional disorders of the liver, but the structural diseases lend themselves more readily to physical examination and medical diagnosis. The fact that the liver is capable of containing a fourth of the blood in the body is sufficient proof of its importance. All the blood passing from the stomach and intestines circulates through the liver, after which it joins the general circulation considerably altered in its composition. Experimental researches show that the liver is concerned in the manufacture of urea or the antecedents of urea.<sup>1</sup> Degeneration or destruction of the hepatic cells is attended by a diminution in the quantity of urea excreted, and the amount of urea in the urine may be taken as a valuable prognostic guide in many hepatic disorders. The metamorphosis of the products of digestion in the course of their elaboration into urea is therefore one of the functions, probably the chief function, of the liver. Another important function of the liver is the manufacture of glycogen; the third and least important is the secretion of bile.

#### PART A. SYMPTOMATOLOGY.

The symptoms due to disorders of the liver are not so clearly defined as those of cardiac or pulmonary diseases. The cardinal symptoms of *structural* disease of the liver are PAIN IN THE HEPATIC REGION, JAUNDICE, and a group of symptoms due to PORTAL OBSTRUCTION, which include Ascites. When the liver cells become gradually destroyed, as in cirrhosis, serious disturbance of the general health ensues, and in the later stages of that and of some other hepatic disorders, LETHARGY passing into coma supervenes.

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<sup>1</sup> If blood containing no urea be passed artificially through the liver, it is found that the effluent fluid contains urea, and therefore it must have been added to it in the liver. Again, if the portal vein be connected to the jugular vein, thus shutting out the liver from the general circulation, the formation of urea ceases. Solnikoff and others, see Alexander Hill's "Physiologists' Notebook," p. 82. Cambridge. 1893.

The chief symptom of *functional* derangement of the liver is, according to Murchison,<sup>1</sup> an excess of lithates in the urine, LITHURIA, consequent on an excess of lithic or uric acid in the blood (lithæmia). How far lithæmia is really due to hepatic disorder is still a debated point (§ 251); it may certainly arise in other ways. Functional derangement of the liver is always attended by DEPRESSION, which may amount to hypochondriasis, and vague DIGESTIVE DISTURBANCES.

§ 232. **Pain and Tenderness over the Liver** is very marked in PERIHEPATITIS and any other condition in which the *capsule* is involved; sometimes radiating upwards towards the right scapula. The onset of pain in the course of a liver complaint may therefore be of considerable importance; for example, in hydatid of the liver, the natural course of which is painless, it would point to a danger of rupture of the cyst. When the upper surface of the liver is involved, the pain is very often *referred to the right shoulder*; it is, indeed, a symptom of phrenic (diaphragmatic) irritation. The most severe form of pain, however, is that which occurs in connection with the passage of GALL-STONES (*biliary colic*). In a considerable number of hepatic disorders, pain may be completely absent. There is, however, in many cases of marked disease or enlargement of the liver a feeling of weight or fulness in that region, accompanied by an inability to lie on the left side.

Hepatic pain may be *simulated* by Pleurodynia (rheumatism of the intercostal muscles), Intercostal Neuralgia, Pleurisy, Dyspepsia, and various gastric conditions, and by Intestinal or Renal Colic.

§ 233. **Jaundice** is the term applied to the yellow pigmentation of the skin and other tissues due to the non-elimination of bile; it appears first in the urine, in which bile pigments and acids may be detected (§ 284), next in the conjunctivæ, then in the skin universally and uniformly.

FALLACIES. The yellow coloration of the conjunctivæ differentiates jaundice from all similar pigmentations of the skin. (1) Excess of *sub-conjunctival fat* may simulate jaundice, but this is readily distinguished by its unequal distribution. (2) The *sallowness* of the skin in chlorotic young women is easily distinguished from jaundice by the absence of pigment from the conjunctivæ, and the absence of bile from the urine. (3) The *cachexia* of carcinoma, malaria, and certain other forms of visceral disease, is differentiated in the same way. (4) The *bronzing*

<sup>1</sup> "Clinical Lectures on Diseases of the Liver" Longmans & Co. 2nd ed. London. 1877.

of the skin in Addison's disease is hardly likely to be mistaken for jaundice. (5) *Santonin* and *rhubarb*, administered internally, colour the urine, but do not give the reaction for bile in that fluid.

*Symptoms accompanying jaundice.* (1) Flatulent dyspepsia, and a bitter taste in the mouth. (2) Pruritus which may be very troublesome in some cases; eruptions, such as xanthelasma, are less common. (3) The temperature, as a rule, is subnormal, and the pulse slow; (4) general debility and emaciation ensue in prolonged cases; (5) head symptoms, such as delirium and coma, may appear towards the end; and xanthopsia, *i.e.*, a yellow vision, is sometimes present.

The *causes of jaundice* may be divided, on both clinical and pathological grounds, into two great groups, (a) those producing **obstruction** of the smaller or larger biliary passages, and (b) those in which no obstruction can be made out, when the condition is called **non-obstructive** jaundice.<sup>1</sup> Clinically, obstructive jaundice is distinguished from non-obstructive by the *colour of the stools*, which are pale, slate, or clay-coloured, from the absence of bile from the intestinal canal in the former variety.

Following modern pathological teaching, these two groups are now known also by the names **Hepatogenous** and **Hæmatogenous** respectively. In the former the causation depends on some lesion in or near the liver: while the latter depends upon some defective action of the blood or tissues, such as the deficient transformation of bile acids and pigments into urinary pigments (Murchison, *loc. cit.*), or the suppression of the function of the liver cells (*e.g.*, as in malignant jaundice), or when they cannot keep pace with excessive hæmolysis (blood destruction), as in certain fevers. There are four separately named forms of jaundice—CATARRHAL JAUNDICE (§ 241), EPIDEMIC JAUNDICE, or Weil's disease (§ 242), MALIGNANT JAUNDICE, Icterus Gravis, or Acute Yellow Atrophy of the Liver (§ 249), and ICTERUS NEONATORUM (§ 234).

(a) **Obstructive jaundice** (Hepatogenous J.) may be produced in three ways—Obstruction within the duct, disease in the wall (II. and III. below), or pressure outside the bile-ducts.

I. FOREIGN BODIES within the duct, such as (1) gall-stones and inspissated gall; (2) hydatids, round worms, distoma,<sup>2</sup> and other parasites; (3) foreign bodies from the bowel.

II. CATARRHAL INFLAMMATION of the bile ducts, usually spreading

<sup>1</sup> Murchison's classification, somewhat modified, is the one adopted here. *Loc. cit.*

<sup>2</sup> If the patient has resided at the Cape, or other country where the parasite is found, the stools may be examined for the eggs of the *Distoma hepaticum*. In these cases the eggs are very plentiful, and contained in every stool. Besides jaundice, which is often of an intermittent character, the patient complains of a dull aching in the hepatic region, and gastric disturbance.

from the duodenum and stomach. This, which is known as CATARRHAL JAUNDICE, is one of the commonest forms of jaundice (§ 241).

III. STRICTURE, or obliteration of the duct owing to (1) congenital absence; (2) perihepatitis; (3) cicatrization after ulcer of duodenum; (4) ulceration of the bile duct, which may produce obstruction by the swelling around or lead to stricture; and (5) spasmodic stricture (?).

IV. TUMOURS pressing on the duct, such as (1) cancer and other tumours of the liver; (2) enlargement of the glands in the transverse fissure of the liver; (3) tumours of the stomach, pancreas, kidney, great omentum; (4) faecal masses in the intestines; (5) pregnant uterus; (6) ovarian tumours; and occasionally (7) tumours growing from the walls of the ducts.

(b) **Non-obstructive jaundice** (Hæmatogenous J.) may arise from two groups of causes—

I. POISONS IN THE BLOOD, such as occur in (1) cirrhosis of the liver (occasionally); (2) pneumonia, the acute specific fevers, especially tropical fevers, yellow fever, relapsing fever, and pyæmia, (3) animal poisons, such as ptomaines or snake-bite; (4) chemical poisons, such as phosphorus, mercury, antimony, arsenic, copper, chloroform, and ether; and (5) acute yellow atrophy of the liver.

II. IMPAIRED OR DERANGED INNERVATION, in which case the jaundice may come on very suddenly, such as (1) severe mental emotion; (2) concussion of the brain; (3) deficient oxygenation of the blood; (4) excessive secretion of bile; (5) excessive absorption of bile, such as arises in protracted constipation.

To *diagnose which of the causes* of jaundice is in operation:—1, if possible, EXAMINE THE FECES, which are slate or clay-coloured in obstructive, and of normal colour in non-obstructive jaundice. But it must be remembered, as possible fallacies, that in obstructive jaundice the coloration of the skin persists for a time after the obstruction has been removed; that the fæces may become stained if mixed with urine; and that the bile duct may be only partially obstructed, and enough bile may thus escape to colour the fæces.

2. Inquire as to the HISTORY OF THE ATTACK. Jaundice coming on suddenly, especially in a middle-aged female patient previously



in good health, almost invariably indicates obstruction by gall-stones (rare cases of nerve shock excepted). Jaundice coming on slowly, and ultimately becoming intense, is very generally due to a tumour pressing on the hepatic duct. A well-marked jaundice persisting some weeks is almost certainly obstructive. A history of previous temporary attacks points in adult life to gall-stones, in youth to "catarrhal jaundice."

3. EXAMINE THE HEPATIC REGION carefully. If the liver is enlarged, cancer is the most probable cause; interstitial hepatitis less commonly. If ascites be present, the diagnosis rests between cancer and cirrhosis.

4. Inquire as to PAIN AND CONSTITUTIONAL SYMPTOMS. Pain of a spasmodic and severe character accompanies jaundice due to gall-stones and cancer. It is more constant and gnawing in character in congestion of the liver and catarrh of the bile-ducts. The temperature is not often elevated, but it may be so in catarrhal jaundice, jaundice due to poisons in the blood, pyæmic hepatitis, tubercular affections, and local pus formations, such as inflamed hydatid. Cerebral symptoms are very rarely present, except a fatal termination is at hand, unless the jaundice occurs in the course of pneumonia, fevers, or in that rare disease, acute yellow atrophy of the liver.

The *prognosis* and *treatment* of jaundice depend on its causal diseases (*q.v.*). The disappearance of bile from the urine indicates that the attack is coming to an end; though it may be some weeks before the skin clears. The flatulent dyspepsia and many of the concurrent symptoms may be relieved by the administration of ox-gall (gr. 5—10 or more) with meals, together with carminatives. The itching of jaundice is often a most troublesome symptom, but it can generally be relieved by pilocarpine.

§ 234. **Icterus Neonatorum** is a mild transitory form of jaundice which affects a very large number (estimated by various observers at from 70 to 90 per cent. of all children born<sup>1</sup>) of new-born infants. It appears usually on the 2nd or 3rd day of life, is not generally very intense, and rarely lasts longer than 1 or 2 weeks. The feces are normal in colour, and apart from the jaundice the infant presents no other symptoms. The cause of the condition has been the subject of considerable debate, but the question is almost entirely an academic one, and the reader is

<sup>1</sup> These statistics were taken from hospital cases which were placed in circumstances where a better light probably resulted in the detection of the slightest tinging of the skin.

referred to systematic works or to a publication by Runge.<sup>1</sup> The treatment, if any is required, is the same as that for catarrhal jaundice.

A *severe form* of the same condition, sometimes erroneously called Icterus Gravis Neonatorum, occurring during the first week of life, may be due to (1) congenital stricture of the bile-ducts by syphilitic perihepatitis; (2) congenital absence of the duct; (3) septicæmia; (4) Winckel's disease (an epidemic form); or (5) acute fatty degeneration of the new-born (Buhl). The 1st and 2nd are diagnosed by the intensity of the jaundice and the absence of bile from the fæces; the remainder present other symptoms, such as hæmorrhages, purpuric spots, and (4 and 5) cyanosis.

#### PART B. PHYSICAL EXAMINATION.

The liver lies chiefly in the right hypochondrium; the left lobe extends across the epigastrium above the stomach, into the left hypochondrium. The gall-bladder lies below, in contact with, the liver, and is situated under the 9th right costal cartilage (see Figs. 62 and 72).

The routine methods of examination of the liver consist of INSPECTION, PALPATION, and PERCUSSION.

§ 235. **Inspection** locally teaches us but little, as a rule, unless the symmetry of the abdomen as observed from the foot of the bed be altered. However, the presence or the absence of *jaundice* should always be noted in cases of suspected hepatic disease. If slight, it may be noticeable only in the conjunctivæ. Note also if there are venous stigmata in the face or enlargement of the veins of the abdominal wall, such as occur with cirrhosis and portal obstruction.

§ 236. During **Palpation**, the patient should be placed in the recumbent posture, and in order to obtain complete relaxation of the abdominal walls, he may be asked to "let his breath go." If this is not sufficient, the knees should be drawn up and the shoulders supported. Standing on the right side of the patient, place the palmar surface of the hand, previously warmed, on the right side of the abdomen, immediately above the iliac crest, pressing it firmly yet gently inwards. The tips of the fingers should be inclined slightly upwards and inwards, towards the median line, and the *upper margin of the index finger* should be pressed firmly down, working little by little upwards towards

<sup>1</sup> "Die Krankheiten der ersten Lebensstage," Auflage 2, 1893.

the costal margin. In this way the upper border of the index finger, always held perfectly flat, will come in contact with the margin of the organ if it be enlarged. But if it is not enlarged the liver cannot be felt, for it lies altogether beneath the costal margin in the adult. In young children, however, the liver is proportionately larger in all its dimensions, and the lower edge normally protrudes beneath the costal margin. If the liver is enlarged, try to feel its surface by gently dipping the fingers down. Notice if its surface is smooth (as in fatty liver) or nodular (as in cancer), or simply rough ("hobnail"). When there is fluid in the peritoneal cavity this method of "dipping" the fingers (suddenly) is also useful; but in most cases the finger tips only excite contraction of the abdominal muscles and so frustrate our object. The other fallacies of hepatic enlargement are mentioned under *Percussion* (below).

The *gall-bladder*, if enlarged, may be discovered as a round elastic tumour, projecting beneath the 9th rib, at its junction with the cartilage.

**Examination of the liver by the "procédé du pouce"** (Glénard<sup>1</sup>). When the liver is difficult to make out, or ptosis, or a floating lobe, is suspected, this method is said to be very useful. In it the lower border of the organ is examined by the pulp of the l. thumb; the r. hand being used to force the intestinal mass under the liver and so tilt its lower edge forwards. The physician should be seated on the r. of the patient, who lies on his back with the knees drawn up. The l. hand is placed behind the patient's r. loin, and presses it forward, the l. thumb being placed in front, just below the costal margin. Next, place the r. hand on the front of the abdomen, just below the umbilicus, the fingers being directed *obliquely downwards and outwards towards the patient's right groin*. Very firm pressure is made with the r. hand so placed, and a *circular* movement is described, so that the fingers presently point upwards and outwards towards the r. hypochondrium. While the hands are so placed the patient is told to take a deep inspiration, and if the l. thumb be made to slide upwards it will slip over the edge of the organ.

§ 237. **Percussion** should be light, so as to elicit only the superficial or absolute dulness of the organ. In percussing the upper margin, start where there is a good lung note above, and percuss down from rib to rib in the nipple, midaxillary, and scapular lines. Then repeat the process from space to space. In defining the lower edge still lighter percussion should be used, and the examination should proceed from the tympanitic note of the intestine upwards towards the hepatic region. But the more certain method of detecting the lower edge is by **palpation**.

Normally, in the nipple line the *superficial* or absolute *hepatic*

<sup>1</sup> "Les Ptoses Viscérales," Glénard, Paris, 1899, p. 625; see also an article by Sir Frederick Treves, in *The Lancet*, May, 1900.

*dulness* commences two fingers' breadth below the nipple and measures  $3\frac{1}{2}$  to 4 inches, and in a routine examination this is the most important measurement to obtain. The normal boundaries of the liver are given in Fig. 72.

The *lower border* arches upwards just beneath the r. costal margin and crosses the epigastrium; where the hepatic dulness becomes continuous with the cardiac dulness. In the mid-sternal line the dulness extends from  $\frac{1}{2}$  inch above the base of the xiphoid

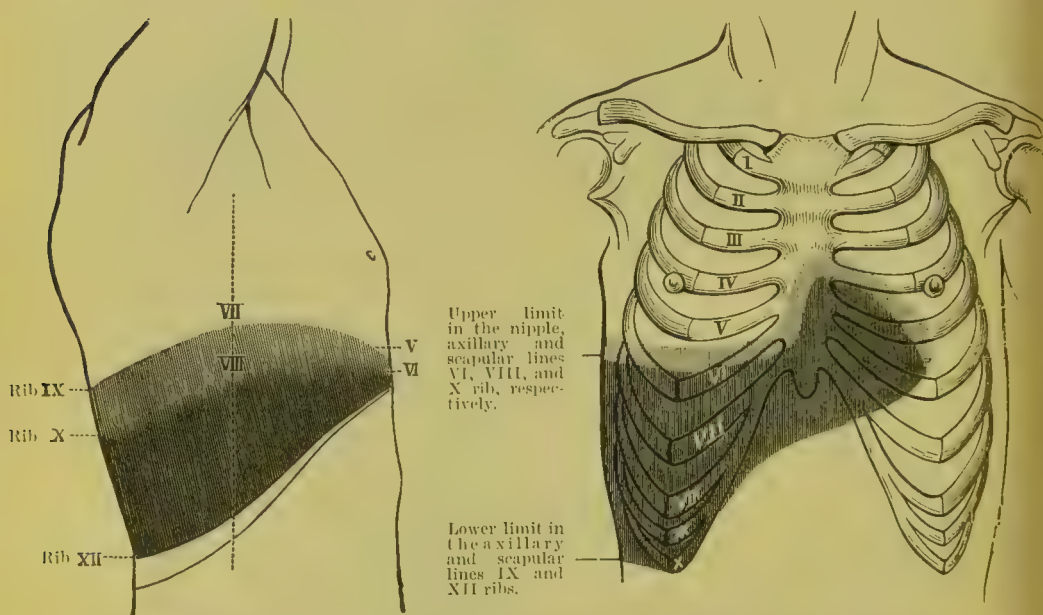


FIG. 72. —AREA OF LIVER DULNESS. The *superficial* (or absolute) dulness corresponds to the deep shading; the area of *deep* (or relative) dulness is larger and includes the lighter shading.

cartilage to about midway between the umbilicus and the xiphoid, where the lower edge may be felt by careful palpation when the abdominal wall is very lax. Thus the *absolute* dulness measures on an average about 2 inches in the mid-sternal line and 4 inches in the nipple line.

These landmarks do not indicate the deep position of the liver, which is more difficult, and in most cases less useful, to determine. But in some cases, such as abscess or hydatid, it is desirable to make out the deep (or *relative*) dulness of the liver, by heavy percussion. The extreme height of the liver, as thus made out, corresponds to the 5th rib in the nipple line, 7th space in the mid-axilla, and 9th space in the scapular line.



LIGHT PERCUSSION BOUNDARY (the one ordinarily used) gives the superficial or absolute dulness—*i.e.*, where the liver is in contact with the ribs.

	Nipple line.	Mid-axillary line.	Scapular line.
Upper margin situated at the ... ..	6th	8th	10th rib.
Extent of dulness in vertical line ... ..	3½	4	3 inches.

HEAVY PERCUSSION BOUNDARY, gives the deep or relative dulness where the liver is covered by lung.

Upper margin situated at the ... ..	5th	7th	9th rib.
Extent of dulness in vertical line ... ..	4	5	4 inches.

FALLACIES. The physician should never feel satisfied with mapping out the liver once only, because the organ may be temporarily affected by many varying conditions, and the *percussion* boundaries by no means always give us a true index. Thus, the lower edge may be masked by the dulness of the stomach after a full meal, by an accumulation of fæces in the colon, or by a thickened omentum. Great rigidity of the muscles, or œdema of the abdominal walls, may also obscure the lower edge of the liver.

The beginner, by *palpating* with his finger tips, which excite muscular contraction, generally fails to make out the lower margin of the liver even when the organ is enlarged. By *percussing too heavily* he fails to get the *absolute dulness*.

Apparent *diminution* of the liver may arise from (i.) distension of the stomach or intestines with gas; (ii.) by contractions of Glisson's capsule, especially on the under surface, giving rise to puckering or distortion of shape anteriorly; or (iii.) emphysema of the lungs, which obscures the *upper* border very much. Great diminution or absolute loss of the liver dulness, owing to gas in the peritoneum, is a diagnostic feature of perforation of the stomach, *e.g.*, from gastric ulcer.

Apparent *enlargement*, when attention is paid solely to the lower edge of the organ, may be due to a *displacement* of the liver downwards (i.) by pleuritic effusion, emphysema or pneumo-thorax; (ii.) intrathoracic tumours; or (iii.) enlargement of the heart or hydro-pericardium. These and other fallacies may arise from paying attention solely to the *lower edge* of the organ; and finally, the liver may in rare cases be dropped or "*floating*," a condition which will be dealt with subsequently. "Riedel's lobe" is mentioned under abdominal tumours.

§ 238. Fluid in the Peritoneum (Ascites) is a frequent accompaniment of some hepatic disorders, and its presence or its absence must always be carefully noted. The methods of investigating this important matter have already been given (§ 185).

Ascites (dropsy of the peritoneum) is one of the **evidences of portal obstruction**, and these are more frequently associated with some disease of the liver than that of any other organ. Sometimes they are the only evidences we have of hepatic disorder.

The SIGNS OF PORTAL OBSTRUCTION are, in the order in which they appear—(1) A liability to attacks of *gastric* and *intestinal catarrh*, as evidenced by irritable dyspepsia, and the vomiting of mucus, streaked perhaps with blood, in the early morning before breakfast. (2) *Hæmorrhage*, sometimes in very large quantity, from the stomach and the bowels. (3) *Hæmorrhoids* may occur in other diseases (§ 227), but they are constantly associated with portal obstruction. (4) Attacks of *congestion of the liver*. (5) Congestion and therefore enlargement of the *spleen*. (6) *Ascites* (see below). (7) *Enlargement of the veins* of the abdominal wall from the establishment of a collateral circulation. (8) *Œdema* of the legs is a secondary and indirect result of the pressure of the ascitic fluid on the large veins within the abdominal cavity. (9) *Albumen* in the urine may arise in the same way, or from concurrent disease of the kidney.

ASCITES, it will be observed, is a late sign of portal obstruction. It has already been fully described (§ 186), and it will be remembered that its *three principal causes* were—Cardiac, Hepatic, and Renal disease. (1) In *cardiac disease* the ascites will have been preceded by dropsy of the legs; (2) in *hepatic disease* the ascites is the predominating feature, though it may be followed by dropsy of the legs; while (3) in *renal disorders* the ascites is only part of a dropsy which is general from the outset. *Cancer of the peritoneum* may also produce ascites, but here the nodules of cancer will probably be felt on palpation and there will be other symptoms of cancer. Ascites may have to be diagnosed from an ovarian or other large abdominal cyst, and from fat in the omentum (§ 183).

Portal obstruction, and consequently ascites, are not present with equal frequency in all diseases of the liver, and in some they are absent. In order of frequency they are as follows:—

(1) CIRRHOSIS, or an increase of the interstitial tissue (usually due to alcohol), is by far the commonest cause of portal obstruction, by producing pressure upon the minute branches of the portal vein within the liver.

(2) In CANCER of the Liver portal obstruction and ascites are fairly frequent, but they are due not so much to the cancer within the liver as to the pressure of enlarged glands in the fissure of

the liver upon the portal vein, or to secondary involvement of the peritoneum.

(3) PERIHEPATITIS may occasionally result in constriction of the portal vein by puckering at the fissure.

(4) SIMPLE CONGESTION of the liver may be attended by hæmorrhoids and gastric catarrh, but rarely by much ascites.

(5) Fatty and Waxy Liver, Abscess, and Hydatid are hardly ever attended by portal obstruction.

The other *causes*, the *prognosis*, and *treatment* of Portal Obstruction have been described under Ascites (§ 186).

In cases of hepatic disease the **urine** should always be tested for *bile* (§ 284), and for urates (§ 293), sometimes for leucin and tyrosin; and the amount of urea may need to be estimated (§ 282).

#### PART C. DISEASES OF THE LIVER.

§ 239. **Routine procedure.** FIRST, Ascertain *what is the patient's leading symptom*. The symptoms of disorder of the liver we discussed in Part A.; *e.g.*, gastric disturbance, pain (or a feeling of weight or discomfort in the hepatic region), or jaundice. If there be severe and paroxysmal pain, turn first to biliary colic (§ 243).

SECONDLY: Learn the *history* of the patient's illness, eliciting the facts in chronological order, and in this way ascertain the important fact whether the disease be *acute* or *chronic*, because disorders of the liver may be conveniently classified into these two groups.

THIRDLY: THE EXAMINATION OF THE LIVER must next be made. The routine method is as follows:—

1. Ascertain whether the liver is *enlarged* or *diminished* (by percussion in the nipple line, and abdominal palpation); and whether there is any *pain*, *tenderness*, or other abnormality.

2. Ascertain whether there be any *fluid* in the peritoneum (§ 185).

3. Ascertain if there is any *jaundice* (§ 233) and examine the *urine* for bile pigments, lithates; and occasionally the diurnal amount of urea.

**Classification.** For clinical purposes, diseases of the liver may be conveniently divided into ACUTE and CHRONIC Disorders.

If the illness is one of long standing, and has come on insidiously, the reader should turn to **chronic diseases of the liver** (§ 250). The **acute diseases** will be first described.

## ACUTE DISEASES OF THE LIVER.

If the illness has come on more or less suddenly, and is attended by considerable malaise or other constitutional symptoms, it is one of the **acute diseases of the liver**, probably—I. ACUTE CONGESTION, II. CATARRHAL JAUNDICE, or III. GALL-STONES. The less common acute diseases are—IV. PERIHEPATITIS, V. ABSCESS, and VI. ACUTE YELLOW ATROPHY.

TABLE XX.—ACUTE DISEASES OF THE LIVER.

	Jaundice.	Enlargement of the Liver.	Ascites.
I. ACUTE CONGESTION.	Not very great.	Slight increase.	Usually absent.
II. CATARRHAL JAUNDICE.	Always present and marked.	Slight increase.	Absent.
III. GALL-STONES.	Very marked in most cases.	May be considerable increase.	Absent.
IV. PERIHEPATITIS.	Absent.	None unless another cause.	Usually absent.
V. ABSCESS OF LIVER.	Generally present.	Moderate and irregular enlargement.	Usually none.
VI. ACUTE YELLOW ATROPHY (v. rare).	Very marked.	Liver diminished in size.	Absent.

I. *The patient complains of PAIN or DISCOMFORT IN THE HEPATIC REGION, the liver area may be INCREASED, slight jaundice and numerous vague DYSPEPTIC SYMPTOMS are present, but there is little or no fever*—the disease is probably ACUTE CONGESTION OF THE LIVER.

§ 240. **Acute Congestion of the Liver.** Clinically, there are two kinds of congestion of the liver, an active or acute congestion, and a passive or mechanical congestion. *Active or arterial congestion* (with which we are now concerned), is usually met with in the form of acute attacks due to dietetic errors; though it may sometimes occur as a more subtle and sometimes latent condition in a subacute or chronic form which eventuates in cirrhosis. *Passive or venous congestion* is due to obstructed venous return (mostly in chronic cardiac or pulmonary disease); it is in the nature of things a chronic process and will be considered under chronic diseases (§ 257).

*Symptoms.* (1) The onset is usually somewhat sudden, after a series of indiscretions in diet, especially in the matter of alcohol.



The patient complains of pain, or a feeling of weight or uneasiness in the region of the liver; and he may be unable to lie on the left side. (2) There is generally a slight but uniform enlargement of the liver, and some degree of tenderness. (3) Slight jaundice is present on the second or third day in the majority of cases, but it is never so intense as in catarrhal jaundice or gall-stones. The fæces are dark in colour, owing to the presence of bile. (4) Certain gastro-intestinal symptoms are present—nausea, headache, furred tongue, a bitter taste in the mouth, and flatulence; the bowels are usually constipated; the urine is scanty, high coloured, and deposits lithates on standing; and there is usually some depression of spirits and irritability of temper.

*Etiology.* (1) By far the most frequent cause is alcoholic excess. Constant indulgence in rich foods containing fats, sugars, and spices, may also produce congestion. (2) Residence in hot climates, especially when associated with malaria and dietetic errors; but many attribute to the climate what is really due to alcohol or faulty diet. (3) Suppression of an habitual discharge, especially bleeding piles or menstruation; and (4) dysentery and febrile states are often accompanied by congestion. (5) Sudden or protracted chill; and (6) injury have been mentioned as causes. An attack of acute congestion may be *predisposed to* by (i.) the presence of chronic congestion (§ 257); (ii.) previous attacks of malaria; (iii.) indolent or sedentary habits.

The *diagnosis* is based upon the occurrence of symptoms of gastro-intestinal disturbance in association with pain and enlargement of the liver. In *perihepatitis* the first named are absent, the pain is much more acute, and syphilis is probably in operation. The diagnosis from the other acute hepatic disorders is given in Table XX. (*ante*). The symptoms of *pleuro-pneumonia* at the onset may include jaundice and the other symptoms of acute congestion of the liver, for which indeed this disease may be mistaken. It is important, therefore, to examine the base of the right lung in all such cases when associated with jaundice.

*Prognosis.* Acute congestion is very apt to recur, especially if the patient continues his dietetic indiscretions. The intervals between the attacks become shorter, and the condition is followed by sub-acute or chronic congestion, and, eventually, cirrhosis. An

attack of moderate severity rarely lasts more than a week or two. Unless a condition of cirrhosis is reached (when enlargement is checked to some extent by the shrinking of the newly formed fibrous tissue), the degree of congestion may be fairly estimated by the amount of enlargement.

*Treatment.* The indications are:—(1) To relieve the congestion of the portal system, and (2) to correct dietetic errors. To relieve the congestion, saline purgatives are especially indicated, such as the sulphates of magnesia, potash, soda, or the bitartrate of potash. Carlsbad, Friedrichshall or Hunyadi Janos water should be taken every morning early, and a full dose of calomel, podophyllin, or pil. hydrarg. at night. In severe cases leeches, or dry or wet cupping in the region of the liver, may relieve the pain considerably. Leeches are sometimes applied to the margin of the anus, but this is not always convenient. Murchison recommended ammonium chloride in doses of gr. xx two or three times a day, to induce free diaphoresis, and diminish the portal congestion and pain (F. 46, 51, 53, and 66 may be useful). Ipecacuanha is in great repute among Indian physicians, and is given in large doses, as in dysentery, xx to xxx grs. every 6 or 12 hours, preceded, half an hour before each dose, by  $\frac{1}{2}$  gr. of opium to prevent vomiting. For the gastric symptoms, alkalies, the alkaline salts, carbonate of magnesia, bismuth, and hydrocyanic acid are useful. Nitro-hydrochloric acid and nux vomica are also used in convalescence. The diet during the attack should be of the simplest, consisting at first solely of two pints of milk a day. Alcohol in any form should be strictly forbidden. As the patient recovers, a simple regimen should be prescribed (§ 214).

II. *The patient, who is YOUNG, has suffered from GASTRO-INTESTINAL DISTURBANCE for some days or weeks, when OBSTRUCTIVE JAUNDICE (i.e., with clay-coloured stools) sets in somewhat suddenly, without local pain, and with little or no enlargement of the liver—the disease is probably CATARRHAL JAUNDICE.*

§ 241. **Catarrhal Jaundice** (Acute Cholangitis) is jaundice due to inflammatory (catarrhal) swelling of the lining membrane of the bile ducts, and the consequent obstruction to the outflow of bile. It is therefore an *obstructive* form of jaundice.

*Symptoms.* (1) The jaundice is usually of sudden onset, though it is preceded for a longer or shorter time by signs of gastrointestinal disorder. (2) The jaundice is often very intense; but in mild cases the degree corresponds to that of congestion of the liver. It generally begins to subside in the course of two or three weeks. If it lasts longer, some other cause (§ 233) should be suspected. (3) The stools are clay coloured, and the urine is dark with bile. (4) Nausea and loss of appetite, flatulence, and constipation are generally present. (5) A feeling of uneasiness or weight in the hepatic region is usually complained of. There may be slight enlargement of the liver, the edge being smooth, firm, and tender. The spleen may be slightly enlarged. (6) There may be slight fever at the commencement, but it usually subsides before the patient is seen; and the pulse is abnormally slow.

*Etiology.* (1) Extension of inflammation from the stomach and duodenum along the bile ducts secondarily to gastric derangement is the most common cause of catarrhal jaundice. (2) It is the by far commonest form of jaundice met with in children and young adults. (3) Exposure to chill. (4) It may be secondary to congestion (§ 240, *ante*) or cancer. (5) Catarrhal jaundice frequently follows the passage of a gall-stone. (6) In the adult, gout or gouty conditions are said to predispose.

*Diagnosis.* Catarrhal jaundice may have to be diagnosed in an old person from *cancer*; but in the latter the jaundice comes on slowly, with pain, and it lasts many months. In *gall-stones* there is biliary "colic" (§ 243). In *congestion* of the liver the jaundice is less marked and the fæces are not clay coloured.

*Prognosis.* The disease is never fatal. It usually terminates in a few weeks, after the gastric disorder has been relieved. The outlook is unfavourable only when catarrhal jaundice complicates other maladies, such as cancer or gall-stones.

*Treatment.* (1) Remove any cause of the concurrent gastro-enteritis, and allay the condition with alkalies, alkaline carbonates, rhubarb, bismuth, etc. (2) A brisk mercurial purge, followed by a saline once or twice a week, helps to relieve the congestion, both of the intestines and the liver. (3) Ox-gall, creasote, or salol, are sometimes useful as intestinal antiseptics. (4) Rectal injections of one or two pints of hot water daily (60 to 90° F.), retained as

long as possible, have been recommended to allay any intestinal irritation. The prescriptions and much of the treatment for Congestion (*supra*) are applicable.

§ 242. **Epidemic Jaundice** (Synon.: Weil's Disease, Septic Jaundice (Fraenkel), Infective or Febrile Jaundice) is characterised by a sudden sharp febrile attack accompanied by severe pains in the limbs, and rapidly followed by jaundice, swelling of the liver and spleen, and nephritis; and ending gradually in recovery about the 10th or 15th day. Clinically it resembles a severe transitory form of catarrhal jaundice occurring epidemically. Cases of what was probably the same disease were first described by Weiss in 1866.<sup>1</sup> In 1886 Professor Weil, of Heidelberg,<sup>2</sup> described four cases of a peculiar form of acute infective disease characterised by jaundice, which most German authorities regarded as a disease not hitherto observed.

*Symptoms.* The malady comes on suddenly with marked prostration, headache, and sometimes delirium. The muscular pains, especially in the legs, are among the most noticeable features, and may obscure the other symptoms. The jaundice appears on the 2nd or 3rd day, reaches a moderate degree, lasts about 14 days, and then disappears gradually. The stools are mostly clay coloured. The liver is considerably enlarged and tender, the spleen enlarged, and the urine contains albumin, epithelial casts and sometimes blood. The temperature reaches 103 or 104° on the 2nd or 3rd day, and begins to fall between the 6th and 9th. Various rashes and occasionally epistaxis have been observed.

*Etiology.* There seems to be no doubt that the disease is infectious, or at any rate epidemic. It has always occurred in an epidemic form, chiefly amongst men between the ages of 15 and 30, and especially working-men. Most epidemics have occurred in the summer months. The nature of the infection is not yet identified, though Jaeger<sup>3</sup> succeeded in discovering an organism, to which he gave the name of bacillus proteus fluorescens, in the urine and organs in several cases. The same observer found that the ducks and geese about the river in which the affected patients had bathed, were subject to a fatal disease with marked jaundice. Dr. Wm. Hunter points out (*loc. cit.*) that a very similar jaundice can be produced experimentally in dogs by toluylendiamin, in whom it also produces swelling of the spleen and liver, and nephritis. In both Weil's disease and poisoning by this reagent, the duodenum is frequently found to be the seat of considerable congestion.

Inferentially, the treatment of this Epidemic Jaundice would appear to resemble that of catarrhal jaundice.

§ 242a. **Chronic cholangitis**, or chronic inflammation of the biliary passages, is not a condition that has hitherto been clinically recognised, but Dr. Morley Fletcher communicated a case of this affection to the Pathological Society of London, on Jan. 15th, 1901.<sup>4</sup> The patient was a child *æt.* 3½, who was admitted suffering from severe dyspnoea and signs of bronchitis. The liver was enlarged but there was no jaundice. The diagnosis of the hepatic condition was not made during life. But after death, 5 days after admission, the liver was found to contain numerous cystic spaces filled with bile-stained debris. The bile ducts were surrounded by newly-formed fibrous tissue, and it was evident that the

<sup>1</sup> "Zur Kenntniss und zur Geschichte der sogenannten Weilschen Krankheit;" Wein. med. Woch. 1890, Bd. xl.

<sup>2</sup> "Ueber eine eigenthümliche, mit Milztumor, Ikterus, und Nephritis einhergehende akute Infectiouskrankheit;" Deut. Archiv. für Klin. Med. xxxix. 1886.

<sup>3</sup> Quoted by Dr. William Hunter in Albutt's "System of Medicine," vol. iv., p. 95.

<sup>4</sup> See Report of the Pathological Society in *The Lancet*, Jan. 19, 1901.



cysts were dilated portions of the biliary passages. There was no history of syphilis, and the origin of the lesions was obscure, but the general consensus of opinion was that the case was one of chronic cholangitis; the child having probably died before jaundice could be developed, which, it would seem, would be a necessary clinical attendant in such cases should they live long enough.

III. *The patient, usually an elderly female, is suddenly seized with PAROXYSMIS OF SEVERE PAIN in the hepatic region, and in the course of 12 to 24 hours she becomes JAUNDICED, the stools becoming clay coloured—the case is an attack of BILIARY COLIC.*

§ 243. **Gall-stones and Biliary Colic.** Gall-stones are concretions of bile or of some of its constituents, which form in some part of the biliary passages, most commonly in the gall-bladder. CHOLELITHIASIS is the condition in which gall-stones are developed. When moving along, or impacted within, any of the ducts they give rise to Biliary Colic.

GALL-STONES may vary in size from particles hardly larger than a sand-grain to the size of a golf-ball. They are round or oval in contour, when they are solitary. It is important to notice the presence of facets or flattenings of their surface, caused by the pressure of one against the other, because this indicates that there has been more than one stone in the gall-bladder or bile-ducts. Their colour varies from a yellow to a dark-brown, and their chief physical characteristics are the smooth "soapy" surface, the ready way in which they crumble between the thumb and finger (though sometimes they are very hard), and their lightness as compared with renal calculi. They consist chiefly of cholesterin mixed with bile pigment. Strong sulphuric acid when added to cholesterin crystals gives a ruby red at the junction. But the *appearance* of the crystals is the most characteristic feature about them, consisting as they do of rectangular plates broken by irregular rectangular fractures (Fig. 73).

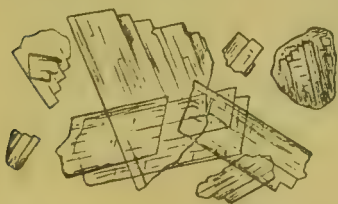


Fig. 73.—Cholesterin Crystals. Microscopic appearance presented by fragments of gall-stones in the feces.

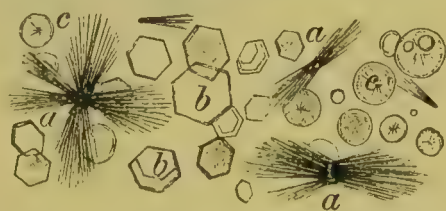


Fig. 73A.—a, TYROSIN, in bundles of needle-shaped crystals, and c, LEUCIN, spherical crystals with concentric markings, found in the urine in rare cases of acute yellow atrophy of the liver. b, CYSTIN (clear 6-sided plates), is a rare urinary deposit associated with some renal (e.g., calculous) and hepatic disorders under conditions not at present understood.

**Biliary Colic.** Symptoms may be absent when the stone is at rest, but when it begins to move (i.) the pain is agonising; it starts in the epigastrium and shoots into the right hypochondriac region towards the spine and up to the right shoulder, but never passes

downwards. The paroxysm is usually so severe that the patient is in a state of partial collapse, with vomiting, hiccough, subnormal temperature, and a quick weak pulse. Sometimes there is a rigor, and the temperature rises a few degrees. Between the paroxysms of acute pain there is a constant dull aching and tenderness over the hepatic region. The attack lasts from a few hours to a few days. (ii.) The liver may be enlarged; and if a stone becomes impacted in the hepatic duct the enlargement may be considerable. (iii.) Jaundice usually appears 12 to 24 hours after



Fig. 74.—The STOMACH AND DUODENUM opened to show the ducts in connection with the Liver and Pancreas.

the paroxysm, and lasts from a few days to a few weeks; it is most intense when the stone is impacted in the common duct.

The *symptoms* which arise vary somewhat with the *position of the gall-stone* (Fig. 74). Thus:—(i.) If a stone is impacted in the *common duct*, there is biliary colic, marked jaundice, and a distended gall-bladder, and if the impaction continue for any length of time the liver becomes enlarged. (ii.) If a gall-stone be impacted in the neck of the gall-bladder (*i.e.*, in the *cystic duct*), biliary colic is present without jaundice. In time the gall-bladder may be distended with mucus, and form a definite abdominal tumour. (iii.) Stone impacted in the *hepatic duct* is rare. It causes biliary

colic and jaundice, but the gall-bladder is not distended. (iv.) Stones occasionally form in the radicles of the hepatic ducts, and give rise to indefinite symptoms, sometimes without pain, and usually without jaundice. (v.) Sometimes small particles of cholesterolin (biliary sand) in the *gall-bladder* may give rise to recurring paroxysms of pain, unaccompanied by any other symptoms, which defy diagnosis.

*Diagnosis of biliary colic.* It is distinguished from the two other forms of colic in Table XV., § 173. The severity of the pain and its paroxysmal character usually distinguish it from all other acute diseases of the liver. Pseudo-biliary colic is sometimes met with in nervous women. The diagnosis from *cancer* of the liver may be very difficult. Both occur at the same age, and both cause jaundice; further, cancer may follow after years of trouble from gall-stones. In cancer, the jaundice comes on and steadily gets more and more intense. It must be remembered that in some cases gall-stones are passed without colic, but with jaundice; consequently, *recurring attacks* of jaundice in an elderly woman should lead one to suspect gall-stones. In all suspected cases the stools should be carefully examined for stones. *The presence of ascites* points to cancer, for it rarely exists long without the effusion of fluid into the peritoneum.

The symptoms of GALL-STONES AT REST in the GALL-BLADDER (CYSTO-CHOLELITHIASIS) are often very obscure, and occurring as they do in elderly females are very apt to be mistaken for cancer. (1) Enlargement and tenderness of the gall-bladder can generally be made out below the 9th c.c., unless it is obscured by adhesions, or by Riedal's lobe, a local hypertrophy of one lobe of the liver sometimes associated with chronic cholelithiasis (see § 189). This and the inefficacy of quinine are the only means we have of distinguishing the condition from ague. But on the other hand the enlargement of the gall-bladder may be mistaken for cancer, from which it can be distinguished only by the long duration of the illness. (2) Attacks of "biliary fever" —*i.e.*, "chills," or shivering with slight rises of temperature of a malarial type — at intervals for months or years are perhaps the commonest complaint. (3) Local pain or discomfort is not always present, but like (1) it may from time to time be produced or aggravated

by exertion. (4) For the rest, the symptoms are negative—no jaundice, ascites, or other symptoms—only a condition of general ill-health, due to septic absorption from the ulcerating and irritated gall-bladder. (5) In the event, the stones may on rare occasions become encysted, but far more often ulceration, perforation, abscess, and fistula result, unless the surgeon intervenes.

*Etiology.* (i.) Gall-stones are much commoner after than before 50 years of age; (ii.) are much more common in women than in men; and (iii.) in stout persons of sedentary habits, who consume a diet rich in fat and sugar. (iv.) There is frequently a history (family or personal) of gout, asthma or migraine, and Sénac found 98 out of 128 cases were associated with urinary gravel. They are less common in hot countries. When gall-stones are already formed in the gall-bladder, an attack of biliary colic is often determined by a sudden strain or an overloaded stomach.

*Course and Prognosis.* The prognosis as to recovery from an attack of biliary colic is excellent, but recurrence may be expected. A stone usually forms in the gall-bladder and becomes impacted for a time in the neck of the cystic duct, giving rise to biliary colic without jaundice. It then passes down the common duct, where it causes jaundice. This rarely lasts more than a few weeks, but rare cases have been reported where it lasted two years. Impaction for any length of time leads to consequences, which may be classified thus:—(i.) *Ulceration* of the ducts, with pyrexia, or abscesses of the liver and bile-ducts, and consequent sub-acute pyæmia; (ii.) *perforation* into adjacent tissues, leading, for example, to fatal peritonitis; (iii.) inflammation and *abscess* of the gall-bladder, which may open externally, perforate into the peritoneum, or ulcerate into the intestines; (iv.) formation of *fistula* between the gall-bladder and the colon or duodenum, through which stones can pass of such a size that they may cause intestinal obstruction.<sup>1</sup>

*Treatment.* (a) *During the attack* anodyne treatment is called for. Opium or a hypodermic of morphia and atropine should be given (F. 25). Chloroform inhalations are used in severe cases. Other drugs recommended are chloral, spt. ætheris, antipyrin, and ext. belladonnæ. Of late years olive oil, in doses of at least six

<sup>1</sup> Large gall-stones may gradually ulcerate through from the gall-bladder to the duodenum, in some cases almost without symptoms. Murchison collected some 34 of such cases where the gall-stone was large enough to give rise subsequently to intestinal obstruction.



ounces, has been strongly recommended as causing rapid passage of the stone into the duodenum, but the author's experience does not support this. Hot water, with one drachm sodii bicarb. to the pint, may be tried. If the patient is put into a warm bath (100° F.), and kept there till he shows signs of weakness, an attack of pain may be warded off. Hot turpentine stupes may give relief.

(b) *Between the attacks* the habits of the patient must be corrected. Wine, beer, and sugar must be avoided. A prolonged course of alkalies, and sodii salicyl., or of such mineral waters as Vichy and Carlsbad, is advisable. Turpentine (℥ x), in capsules, is said to aid the alkaline treatment. The treatment for Congestion (§ 240) is applicable. Surgical treatment is necessary whenever there is suppuration, or when the gall-bladder remains distended, or when the common duct is blocked.

§ 244. **Diseases of the Gall-bladder** are chiefly manifested by pain and enlargement or swelling of the gall-bladder, which first appears just beneath the tip of the ninth rib. The fallacy of Riedal's lobe, see Abdominal Tumours, §§ 188, 189. Mayo Robson<sup>1</sup> gives the following classification of diseases of the Gall-bladder and Bile-ducts:—

A.—**Catarrhal Inflammations**:—(a) ACUTE CATARRH (which corresponds to catarrhal jaundice, § 241); (b) CHRONIC CATARRH. B.—**Suppurative inflammations**:—(a) SUPPURATIVE CATARRH, which may consist of—(α) Simple empyema, and—(β) Suppurative cholangitis; (b) ULCERATION, PERFORATION, and STRICTURE of the gall-bladder and bile-ducts; (c) ACUTE PHLEGMONOUS INFLAMMATION and gangrene of the gall-bladder.

CHRONIC CATARRH of the gall-bladder presents symptoms resembling gall-stones within it (*vide supra*), but there is less pain, very slight jaundice, and no tenderness or pressure over the region of the gall-bladder.

SIMPLE EMPYEMA of the gall-bladder, without involvement of the hepatic ducts, is nearly always due to gall-stones. There is swelling, with continual localised pain and tenderness; and the abscess may burst in various directions, or point externally.

SUPPURATIVE CHOLANGITIS is practically indistinguishable from pyæmic abscesses (§ 246).

ULCERATION of the gall-bladder is referred to above under gall-stones (§ 243); and PERFORATION is usually a result of the same lesion.

STRICTURE is generally also a consequence of the ulceration following gall-stones, but it may sometimes be due to other lesions (see § 233). The result depends upon the position of the stricture. In the *cystic* duct it leads to distension of the gall-bladder. In the *common* duct it leads *both* to distension of the gall-bladder and considerable enlargement of the liver. It is rare in the *hepatic* duct, where it produces enlargement of the liver.

ACUTE PHLEGMONOUS INFLAMMATION of the gall-bladder (phlegmonous cholecystitis) is a rare affection (Courvoisier collected only 7 cases). It

<sup>1</sup> Allbutt's "System of Medicine," vol. iv.

comes on suddenly, with symptoms resembling perforative peritonitis, and is difficult to diagnose from acute appendicitis. It is usually rapidly fatal.

MEMBRANOUS or FIBROUS CHOLECYSTITIS has been recorded by Dr. H. D. Rolleston, secondary to retained gall-stones.<sup>1</sup>

The less common **Acute Disorders** of the Liver remain to be considered, viz., PERIHEPATITIS, ABSCESS OF THE LIVER, and ACUTE YELLOW ATROPHY.

IV. *The patient complains of PAIN AND TENDERNESS in the hepatic region, aggravated by movement. There is NO JAUNDICE, and all the other symptoms are negative*—the malady is probably PERIHEPATITIS.

§ 245. **Perihepatitis** is inflammation of the capsule of the liver, which becomes opaque and thickened, and by its contraction may lead to considerable distortion of the shape of the liver.

*Symptoms.* (i.) The disease sets in suddenly, with pain in the hepatic region, radiating to the shoulder. There is tenderness increased on movement, pressure, or cough. (ii.) Fever is absent as a rule, and the patient may appear to be in his usual health. (iii.) Friction rub may be felt or heard. (iv.) Unless some other disease is present, there is no jaundice or portal obstruction. (v.) The history of a *cause*, especially *syphilis*, is usually present. It is sometimes part of an inflammation of the liver itself, as an abscess, tumour, or cirrhosis. Sometimes the inflammation extends from adjacent organs, as pleurisy or gastric ulcer, or it may be part of a general peritonitis. Perihepatitis occasionally complicates acute or sub-acute rheumatism.

*Diagnosis.* The characteristic pain and the absence of jaundice differentiate it from many other liver diseases. The conjunction of syphilis is also very characteristic. Cases of cysto-cholelithiasis (p. 439) or gumma of the liver may at times be mistaken for perihepatitis.

*Prognosis.* Simple cases tend to recover. In cases which have lasted for a long time a certain amount of cirrhosis of the liver ensues. Portal obstruction may ultimately result from puckering at the fissure: and considerable distortion of the liver may result in the same way.

*Treatment.* The diet must be spare, and the patient must be kept warm. Salines are given with blue pill and rhubarb. Externally, hot fomentations and poultices give relief, and if the pain is severe leeches are recommended. The cause when known must be treated, *e.g.*, syphilis or rheumatism, with potas. iodidi.

V. *There is ENLARGEMENT of the liver, accompanied by PAIN and tenderness, and the boundaries of the area of dulness are IRREGULAR; there are SHIVERINGS, SWEATING, and INTERMITTENT PYREXIA*—the disease is ABSCESS OF THE LIVER.

§ 246. **Abscess of the Liver** (Hepatic or Subphrenic). Solitary or multiple collections of pus may occur in the liver, due to absorption of septic material, or more rarely to suppuration of pre-existing morbid

<sup>1</sup> *The Lancet*, Report of Path. Soc., Lond., May 24, 1902.

deposits such as hydatids or gummata.<sup>1</sup> Abscess of the liver is so common after residence in warm climates that it is spoken of as "Tropical" abscess. Usually the tropical abscess is solitary, and pyæmic abscesses multiple, but this is not an absolute rule.

*Symptoms.* (i.) The onset is usually *acute*, with pain and tenderness of the liver; accompanied perhaps by a dry cough, with shallow respiration and digestive disturbance. The pain is affected by respiration, and is worst when the patient lies on the l. side. (ii.) The liver is enlarged; and the enlargement may extend downwards; or upwards even to the nipple. There may be fluctuation. (iii.) More or less jaundice is present as a rule. (iv.) Constitutional symptoms are marked. There is usually high fever, continuous at first, then with increasing oscillations. Rigors and sweats are common. Later on the patient falls into the typhoid state, with emaciation, vomiting, diarrhœa and delirium.

Besides the acute type just described there is an *asthenic* variety, with insidious onset, general failure of the health, and periods of continuous or intermitting fever followed by intervals of apyrexia resembling ague. Cough and dull aching over the liver and in the right shoulder are generally present from the beginning.

*Diagnosis.* (i.) The pain and pyrexia distinguish abscess from *hydatid* (when not in a suppurating condition). (ii.) A distended and *inflamed gall-bladder* is recognised by a history of gall-stones; and its outline may be palpable on examination. (iii.) Abscess is often mistaken for severe *ague*. But ague is amenable to quinine, the elevations of temperature are periodic, and each paroxysm has 3 stages. (iv.) A hepatic abscess may be diagnosed from other swellings of the liver by exploratory aspiration giving the reddish "anchovy sauce" coloured pus which is distinctive.

The insidious cases of liver abscess are always difficult to diagnose, and where health is deteriorating, with obscure pyrexial conditions from time to time, almost every general or local inflammatory disorder may be suspected before liver abscess. Manson advises the physician in the tropics to suspect liver abscess in all obscure abdominal cases with evening rise of temperature. A low form of pneumonia at the base of the right lung so frequently accompanies liver abscess that its presence is an important aid to diagnosis in obscure cases. On the other hand, cases have occurred where medical men, diagnosing abscess, have explored the enlarged livers of *leucocythæmia* and *pernicious anemia*. This mistake may be avoided by examining the blood before resorting to puncture.

*Etiology.* (i.) Hepatic abscess may arise by the suppuration of a pre-existing tumour, such as hydatid or gumma or the ray fungus. The general opinion is that gummata of the liver do not suppurate; yet they suppurate elsewhere, as in the skin. (ii.) Ulceration of the biliary passages, as from gall-stones or ulcerations of the alimentary canal. dysenteric or simple ulcers, are potent causes of the multiple or "pyæmic abscesses." (iii.) Occasionally operations on the rectum are followed by abscess of the liver. (iv.) Injury to the liver. (v.) Pyæmia. (vi.) The chief form of abscess in warm climates is the large and solitary *tropical abscess* which is associated with dysentery. As many patients with dysentery never develop liver abscess, it is now believed that while dysentery is the predisposing cause, there must also be some other agency as a determining cause. That hepatic congestion plays some part is borne out by the fact that whereas abscess rarely follows dysentery in women, children or natives, it is the usual sequence of dysentery in men who have indulged in alcohol and highly-spiced foods.

<sup>1</sup> Hydatid cysts and gummata very rarely undergo suppurative changes, but I have on several occasions found in the dead-house indisputable evidence that both of these may occasionally do so.

*Prognosis.* 1. The case mortality varies from 57 to 80 per cent. Death usually takes place in three weeks in cases with multiple abscesses. The pyrexia increases, and the patient dies in the typhoid state. 2. Solitary abscess may lead to death in a month, or the patient may live for one or two years, with obscure symptoms as described above. (i.) The abscess may burst into the peritoneum, pericardium, or alimentary canal, with a fatal issue, or it may open externally and gradually recover by free discharge. (ii.) Frequently the abscess, especially a "tropical" abscess, bursts into the right lung. The patient develops a severe cough, with signs of consolidation of the right lung base, and the abscess contents are brought up as a red-coloured sputum. Recovery may result, or the continued discharge may lead to death from exhaustion or lardaceous disease.

*Treatment.* When the evidence points simply to acute inflammation of the liver, before the temperature leads one to suspect pus formation, anodyne treatment, such as cupping and hot poultices, are employed. Ammon. chlor. (gr. xx t.i.d.), or ipecacuanha (if dysentery be present), are the best drugs. Saline purgatives, spare diet and absolute rest in bed are necessary. As soon as an abscess is suspected exploratory puncture must be performed; at least six punctures should be made before abandoning the attempt to find pus. If an abscess is discovered free drainage must be established. Dr. Manson advises the use of a large trochar through which a drainage tube is inserted.<sup>1</sup>

§ 247. *Subphrenic Abscess.* Subphrenic abscess (abscess beneath the diaphragm) occurs either after injury, or in the course of disease such as appendicitis or duodenal ulcer from septic absorption. When it is on the right side it is often known as *suprahepatic abscess*.

The *symptoms* are much the same as those of tropical liver abscess. When occurring above the r. lobe, the liver dullness is continued up in the axilla, perhaps as far as the level of the nipple, and is convex, or dome-shaped, upwards. The base of the right lung shows signs of congestion, and there are evidences of pleurisy at one or both bases.

*Etiology.* In men the most common causes are appendicitis and ruptured duodenal ulcer; in women, gastric ulcer. Other causes are extension of hepatic abscess, empyema perforating the diaphragm, extension of kidney or pelvic abscess, and local tubercular or (rarely) cancerous processes.

*Diagnosis.* In a right-sided *empyema* of the chest the upper border of the dullness, when continuous with that of the liver, is concave, being higher towards the spine. In *hepatic abscess* the liver is tender and enlarged below the costal margin, but it is often impossible to distinguish subphrenic from hepatic abscess. A variety containing air so greatly resembles pneumothorax that it is called *pneumothorax subphrenicus*. It is usually due to perforated gastric ulcer or abscess of the lung.

The *prognosis* is fair if surgical treatment is carried out thoroughly and in time.<sup>2</sup>

§ 248. *Actinomycosis of the Liver* is a condition which may be mistaken for abscess of the liver. It is due to the absorption of the ray fungus from the intestines, and starts as one or more foci in the liver substance which slowly enlarge into spherical masses, and which may undergo suppuration, though the frequency of this latter is debated.

The symptoms consist of vague uneasiness referable to the liver, with gradually increasing enlargement—at first uniform, later on, unequal, the organ becoming prominent in one place. Exploration with trochar may yield no results; but if the tumour is laid open the characteristic greenish fluid with pink specks is obtained in which the ray fungus is found which clinches the diagnosis.

VI. *The patient is a pregnant woman, and the illness has been ushered in by deep JAUNDICE and PROFOUND CONSTITUTIONAL SYMPTOMS. The disorder is ACUTE YELLOW ATROPHY OF THE LIVER.*

§ 249. *Acute Yellow Atrophy* (Malignant Jaundice, Icterus Gravis<sup>3</sup>) is a disease characterised by intense jaundice and cerebral symptoms, extensive necrosis of the liver

<sup>1</sup> *The Lancet*, May 17, 1902. Report of Med. Chir. Soc.

<sup>2</sup> Cantlie, *B. M. J.*, vol. ii., 1899, p. 647.

<sup>3</sup> Icterus Gravis is a term used sometimes in a generic sense for any very severe jaundice tending to a fatal issue. Acute yellow atrophy is, according to modern researches, only one form of it (see Discussion at Clin. Soc., Lond.: *The Lancet*, December, 1900).



cells with rapid diminution in volume of the organ, often occurring in pregnant women, and usually ending fatally.

*Symptoms.* (i.) The premonitory symptoms may be slight, resembling a catarrhal jaundice, with in some cases a degree of pyrexia. There is some tenderness over the liver, which becomes marked in the later stages of the disease. (ii.) In a few days or weeks severe symptoms set in, with deepened jaundice, headache and delirium, and the patient passes into the typhoid state. (iii.) Hæmorrhages occur from the stomach, bowel and bladder, and there may be petechiæ under the skin. (iv.) Fever is usually absent during the course of the illness, but at the end there may be high fever. (v.) With the onset of the severe symptoms the liver dulness begins to rapidly diminish. The spleen is usually enlarged. (vi.) The urine is characteristically altered, having a marked diminution in uric acid, urea and salts, while leucin and tyrosin are found crystallising out on evaporating a few drops of urine on a slide (Fig. 79).

*Diagnosis.* Acute Yellow Atrophy is not likely to be mistaken for any other liver disease after the acute symptoms set in. Phosphorus poisoning may closely resemble it, but in that condition the liver is enlarged, signs of irritant poisoning precede the onset of the jaundice, and the urine does not contain leucin and tyrosin.

*Etiology.* *Predisposing causes.* (i.) Acute Yellow Atrophy is most common under middle age, though rare in children; and (ii.) in women, especially during pregnancy. (iii.) Dissipation and excesses of any kind are said to predispose. *Ereiting.* It is said that the onset of this disease has often been preceded by severe mental emotion; and malaria, influenza, and other blood poisons have been said to determine its onset. The malady is probably *microbic in origin*.<sup>1</sup>

*Prognosis.* The disease is very fatal. After the severe symptoms set in the patient usually dies in a comatose condition within a week. Pregnant women usually abort.

The *Treatment* is very unsatisfactory. During the preliminary stage the disease is treated as in catarrhal jaundice. Warm baths, diaphoretics and diuretics may be tried.

#### CHRONIC DISEASES OF THE LIVER.

§ 250. **Routine Procedure.**—It will be remembered (§ 239) in the physical examination of a patient suspected to be suffering from hepatic disease that the *first* and most important question to investigate is whether there is *any enlargement of the organ* (by palpation and percussion). (2) The question next in order of importance is whether there is *any pain or tenderness* in the organ, for reasons which will be apparent below. And then (3) is there *any jaundice*? (4) Is there *any ascites*? (5) In every case of suspected liver disease the spleen (§ 261) and the urine should be carefully examined.

The numerous *fallacies* in the alteration of the size of the liver dulness must be carefully studied (p. 429).

**Classification.** By common consent chronic diseases of the liver are divided into those in which the AREA OF DULNESS IS NOT INCREASED, and those in which the AREA OF DULNESS IS INCREASED; and these latter are grouped into painful and painless enlargements.

<sup>1</sup> See *The Lancet*, Nov. 4, 1900, Report of the Path. Soc., Lond.

A. The organ is of **normal** or **diminished** size in—

I. Functional derangement of the liver . . . . . § 251

II. Atrophic (alcoholic) cirrhosis . . . . . § 252

B. The organ is **increased in size**,—a. *Without pain* or tenderness—

I. Hypertrophic cirrhosis . . . . . § 253

II. Fatty liver . . . . . § 254

III. Lardaceous liver . . . . . § 255

IV. Hydatid and other (in England) rare conditions . . . . . § 256

b. *With pain* or tenderness—

I. Chronic congestion . . . . . § 257

II. Cancer of the liver . . . . . § 258

III. Abscess of the liver . . . . . § 246

TABLE XXI.—CHRONIC DISEASES OF THE LIVER.

	Size and Surface.	Pain.	Jaundice.	Ascites.
I. ADVANCED CIRRHOSIS OF THE LIVER ( <i>atrophic alcoholic cirrhosis</i> ).	After slight enlargement it becomes <b>DIMINISHED</b> . Surface irregular (hobnail).	None.	Generally absent, never marked.	A very prominent symptom.
1a. HYPERTROPHIC CIRRHOSIS. Of alcoholic, syphilitic, or biliary origin.	Enlargement may be very great. Surface hard and may be nodular.	Varies.	Varies.	Usually absent.
II. CHRONIC CONGESTION.	Slight enlargement. Surface smooth.	Present, but slight.	Slight.	Usually some.
III. FATTY LIVER.	Moderate enlargement. Surface smooth.	Absent.	Absent.	Absent.
IV. LARDACEOUS or Amyloid LIVER.	Enlargement may be very great. Surface smooth.	Absent.	Absent.	Absent.
V. CANCER OF LIVER.	Great enlargement. Surface uneven.	Severe.	Usually present.	Usually present. <sup>1</sup>
VI. HYDATID LIVER (rare in this country).	Outline of dulness arched or distorted.	Absent unless near surface.	Usually absent.	Absent.

A. In the first group, in which the liver is of **normal** or **diminished** size, there are only two disorders, I. FUNCTIONAL

<sup>1</sup> The presence of jaundice and ascites depends on enlargement of the glands in the fissure, generally considerable in the later stages.

DERANGEMENTS ; and II. ALCOHOLIC CIRRHOSIS ; and these are two of the commonest hepatic disorders met with.

I. *There is no alteration in the size of the liver, but the patient complains of* LETHARGY, *vague digestive disturbances, sleepiness after meals, furred indented tongue, CONSTIPATION, headaches, and there is a frequent deposit of* LITHATES IN THE URINE *on cooling—there is probably* FUNCTIONAL DERANGEMENT OF THE LIVER.

§ 251. **Functional Derangement of the Liver**<sup>1</sup> certainly constitutes one of the commonest of the minor ailments that affect a highly civilised community. Very careful percussion may perhaps detect slight enlargement, but generally, if there is any enlargement present, it indicates congestion, Active (§ 240), or Passive (§ 257).

*Symptoms.* There are 2 manifestations of functional derangement which deserve special notice—constipation and lithuria. 1. The common complaint, “My liver is sluggish,” is often equivalent to saying that the bowels do not act properly. Certainly, *constipation*, attended by pale-coloured fæces, due to a deficiency in the amount of contained bile, is a frequent accompaniment of disordered liver. The amount of bile in the stools is not, however, an absolute guide to the activity of the liver. *Diarrhœa* alternating with constipation, and flatus passed per rectum, may be present.

2. *Lithuria, i.e.,* excess of urates in the urine, which appear when the urine cools as a pink or orange deposit, is evidence, according to Murchison<sup>2</sup> and many of his followers, of defect in the liver function, more especially of that function which is concerned in the disintegration of nitrogenous foodstuffs. Such deposits are met with when the quantity of urinary water is markedly deficient, or when the proportion of proteid in the diet is in excess ; but excluding these causes, there are three pathological conditions with which Lithuria is specially associated—(a) *febrile diseases, e.g.,* ordinary febrile “catarrh,” where the liver cells may become granular, and the whole organ may be enlarged and congested ; (b) *structural diseases of the liver,* especially such as are attended by congestion ; and (c) *functional*

<sup>1</sup> The introductory remarks at the head of this chapter may well be perused in this connection.

<sup>2</sup> *Loc. cit.*

*derangements* of the liver (Murchison). Under these circumstances, which more particularly concern us now, the deposit of lithates in the urine is a "manifestation of a morbid condition of the blood and of the entire system," for which Murchison suggested the term lithæmia. It is due to the presence in the blood, not necessarily of lithic acid, but of numerous partially elaborated products belonging to the chemical series which connects proteid food on the one hand with uric acid and urea on the other. It is accompanied, according to the same authority, by a great variety of symptoms—depression of spirits, irritability, lethargy, a disinclination for work, aching pains in the limbs, headache, vertigo, sleeplessness sometimes, undue drowsiness at others, dyspepsia, palpitation, irregularity of the pulse and high tension, or sometimes enfeeblement of the circulation and general enfeeblement of the body.

3. "Functional derangement of the liver may exist for years without any other symptom than the frequent deposit of lithates, and occasionally lithic acid, in the urine. But if neglected it may ultimately be the means of developing *gout*, structural diseases of the liver and kidneys, or some other serious malady." (Murchison, *loc. cit.*, p. 615.)

4. *Sugar in the urine*, *i.e.*, temporary or permanent glycosuria (diabetes), may in many cases, if not in all, be regarded as a manifestation of functional derangement of the liver; but we are still in the dark concerning the pathology of this morbid state.

It may, however, be assumed that glycosuria might arise in one or more of 3 ways:—(a) imperfect glycogenesis in the liver, the sugar passing through the liver unchanged; (b) increased conversion of glycogen into sugar, which results whenever the circulation through the liver is increased—*e.g.*, by vaso-motor paralysis of the hepatic artery; and (c) diminished destruction of sugar in the blood or tissues.

*Etiology of lithæmia.* Functional disorder of the liver (and consequently lithæmia and the other symptoms above named) may be *secondary* to (a) the continual over-functioning of the organ, (b) to diseases of the alimentary tract, (c) to diseases of the heart or lungs, and (d), as above mentioned, to pyrexia. When *primary*, its principal causes are—(1) *errors of diet*, especially rich, sweet, greasy foods, and alcoholic beverages. Alcohol combined with sugar (*e.g.*, port and other fruity wines) are specially injurious; or taken in the form of undiluted spirit, particularly on an empty stomach, is infinitely more harmful than dilute alcohol at meal times. (2) *Deficient supply of oxygen*, such as deficient exercise,



or confinement in ill-ventilated rooms. (3) *Tropical climates*, especially when combined with indulgence in unsuitable food or alcoholic excess. (4) "Prolonged *mental anxiety*, worry, and incessant mental exertion" (Murchison). (5) Certain *constitutional peculiarities*, for the most part *inherited*, may render one person much more susceptible than another to any of the above causes.

*Treatment of lithæmia.* (1) *Diet* is certainly the most important feature of the treatment. Avoid particularly sugars, fats, and alcohol. All highly seasoned and rich foods, sweets, pastry, butter, and, in severe cases, potatoes and fruits may have to be given up. "In most cases of lithæmia, a diet consisting chiefly of stale bread, plainly cooked mutton, white fish, poultry, game, eggs, a moderate amount of vegetables, and weak tea, cocoa, or coffee answers best; while in others the patient enjoys best health on a diet composed of milk, farinacea, vegetables, eggs, and occasionally fish."<sup>1</sup> Haig's diet for uric-acidæmia, as he terms lithæmia, is still more rigorous,<sup>2</sup> and undoubtedly the quantity as well as the quality of the food must be regulated. There is no doubt that many sufferers from lithæmia take more food than can be dealt with by the liver. It is here that inherited peculiarities play such an important part, for what is too little food for one man may be too much for another. Various dietaries are given in § 214. (2) Abundant exercise in the open air to supply the necessary oxygen is only second in importance to diet. (3) Hydragogue and cholagogue aperients—*e.g.*, the regular administration of salines (Hunyadi, Carlsbad, or Friedrichshall waters) every morning, and calomel once or twice a week (F. 46, 51, and 67).<sup>3</sup> (4) Personally I have found bark and mineral acids (especially nitro-hydrochloric)

<sup>1</sup> Murchison, *loc. cit.*, p. 615.

<sup>2</sup> Dr. Alexander Haig's diet for uric-acidæmia consists of bread 10 oz., oatmeal 2 oz., milk 2 pints, cheese 2 oz., rice 2 oz., vegetables and fruit 12 oz. Vegetables, fruit, and bread may exceed these quantities, but this observer maintains that the nearer a patient adheres to this dietary the less likely is he to suffer from uric-acidæmia, gout, rheumatism, and allied diseases. These diseases he regards as being largely dependent on faulty diet, and especially the consumption of animal food (proteids) in excess, and such as contain uric acid and its antecedents (*e.g.*, xanthin). The foods quite free from these substances are—bread, macaroni, rice and other cereals, potatoes, vegetables, nuts and fruit.

<sup>3</sup> Luff found that the gelatinous Sod. biurate is precipitated in crystalline form on making alkaline a blood serum with sod. bicarb. But if Pot. bicarb is added this action is *delayed*, and the precipitate *less in quantity*. Thus is explained the advantage of Pot. salts in acute and subacute gout, and the use of vegetables which are rich in Pot. salts. In gouty people the blood is not less, but really more alkaline than normal, from excess of soda salts, which hasten the crystalline deposit. *Lancet*, November 18, 1899: "Causation and Treatment of Gout," p. 1361, and *B. M. J.*, vol. ii. (1899), p. 1163. See also Discussion, B. M. A., *Lancet*, vol. ii. (1899), p. 441, and "Uric Acid," by Haig.

and bark, taken shortly before meals, very efficacious in some cases. (5) Among the other drugs, chlorides, iodides, and bromides are recommended by Murchison for the various conditions, as indicated by the symptoms. Opium is contraindicated. If this treatment fails, turn to that of Acute Congestion (§ 240).

II. *The area of liver dulness is diminished, and if the surface can be felt it is HARD AND UNEVEN (hobnail); ASCITES is probably present, but no very distinct jaundice; the spleen is enlarged, and the patient is subject to HÆMORRHOIDS, and HÆMORRHIAGES from the stomach and bowel*—the disease is **ATROPHIC ALCOHOLIC CIRRHOSIS**.

§ 252. **Atrophic Cirrhosis of the Liver**, or, as it is sometimes called, Alcoholic Cirrhosis, Interstitial Fibrosis of the Liver, or Interstitial Hepatitis, consists of a progressive degeneration of the liver cells, with an increase of the interstitial fibrous tissue, leading to portal obstruction, and a shrinkage of the organ. Pathologists are now agreed that the interstitial fibrosis is secondary to the atrophic degeneration of the hepatic cells. Though a microbe has been found by Adami in association with this disease, fibrosis or cirrhosis of the liver must still be regarded as mainly the result of alcoholic excesses, especially the habit of dram-drinking on an empty stomach.<sup>1</sup> Clinically there are two varieties of Alcoholic Cirrhosis — the *Atrophic* form, which is a very common condition, and the *Hypertrophic* form, which is relatively rare. The adjectives have reference to the size of the organ, for whereas the former soon becomes diminished, the latter is enlarged throughout the disease. The hypertrophic form is further distinguished by a tendency to jaundice without ascites; and histologically the fibrosis has a uni-lobular distribution, instead of being multi-lobular as in atrophic cirrhosis.

*Symptoms.* (1) In the early stage of the disease the organ may be enlarged, though rarely much so; but in the 2nd and 3rd stages the liver dulness is diminished. The liver is small, firm,

<sup>1</sup> The experimental administration of alcohol to animals does not produce cirrhosis. Sometimes cirrhosis occurs in children and adults who have taken no alcohol during their life. A microbe has been found associated with cirrhotic livers in cattle. In man a microbe has been found in the cells of 26 livers examined, both of the hypertrophic and of the atrophic form of cirrhosis. The microbe is very small,  $\frac{1}{200}$ th in. lens being required in order to see it. As it has not yet been cultivated it cannot be said whether it is the same as has been found in cattle.—Adami, *Lancet*, June 30, 1898.

fibrous, and hard, and the surface is often nodulated, hence it is known as the "hobnail," or "gin-drinkers'" liver. There is a feeling of uneasiness and weight in the hepatic region. (2) The onset of the disease is very slow and insidious, extending sometimes over years. Gastric symptoms, such as *morning sickness*, and the other symptoms of alcoholic dyspepsia, are alone complained of for a considerable time. These are followed by symptoms of chronic gastritis, debility, and emaciation. The patient's aspect is very characteristic, with dilated *venous stigmata* in the cheeks. (3) Jaundice appears in the later stages of the malady in about 1 out of 3 cases. (4) Symptoms of portal obstruction occur (§ 238), and hæmatemesis is sometimes the first obvious symptom; the spleen becomes enlarged, and ascites (which is present in 80 per cent. of the cases) may be very considerable in amount. (5) In the concluding stages of this disease, when the secreting tissue of the liver is destroyed, the patient falls into a comatose state, with muttering delirium, which resembles uræmia and the typhoid state, except that there is pyrexia in the latter. This precise clinical resemblance is quite in keeping with the fact that the liver takes part in the elaboration of urea; so that when its cells are destroyed the blood becomes charged with a number of nitrogenous products, which cannot be eliminated because they are not elaborated into urea.

*Etiology.* (1) Cirrhosis of the liver is most common between 35 and 60; it is rare under 25. Men are much more frequently affected than women. (2) Alcohol is undoubtedly the most usual cause of atrophic cirrhosis, especially when taken in small quantities, frequently, or when taken *neat on an empty stomach*, the patient perhaps never becoming intoxicated (compare opening remarks and footnote on p. 450).

*Diagnosis.* Cancer of the liver is only difficult to diagnose from cirrhosis in the early stages; but typically it runs a more rapid course, has more pain, and more intense jaundice. The spleen is not usually enlarged in cancer. In *passive congestion* of the liver with ascites there are evidences of a cause, such as heart or lung disease.

*Prognosis.* The disease has a slower and more insidious onset than hypertrophic cirrhosis (below), and is in most cases a more serious condition. If the patient is seen before signs of portal

obstruction supervene much can be done, but if not until afterwards, the prognosis is grave. The outlook is more favourable in patients who are young (under 30), and where the general health is good. *Untoward Symptoms.*—Although cases are reported where restoration to comparative health has occurred after the development of ascites, it remains true that, as a general rule, with the onset of rapid ascites the end is in view, the patient rarely living more than a few months. The question has been raised whether such cases resulting in recovery were not syphilitic (§ 253, 1c). When there is rapid reaccumulation of fluid after paracentesis, and little benefit derived from treatment, the patient tends towards an early death from exhaustion.

*Treatment* in the early stages is practically the same as that employed for chronic congestion of the liver, and chronic gastritis (§§ 257 and 210). The habits of the patient must be corrected, and the diet reduced to the simplest elements; milk should be the staple diet in advanced cases. Alcohol must be completely cut off, and regular exercise taken. A course of salines should be ordered to be taken in the early morning, and rhubarb or mercurial pills at night. Ammonium chloride and iodide of potassium are valuable remedies in the stage of enlargement of the liver. If portal obstruction and ascites have set in, see § 186. Patients sometimes recover after repeated tapplings, which gives time for the establishment of the collateral circulation; and recently surgical measures have been adopted for the artificial production of peritoneal adhesions for the establishment of the collateral circulation.<sup>1</sup>

We now turn to those Chronic liver diseases in which **the area of dulness is increased**. These may be divided into two groups—those WITHOUT PAIN AND TENDERNESS, commencing immediately below. If the enlargement is attended WITH PAIN AND TENDERNESS, turn to § 257.

There are four diseases with **enlargement** of the liver **without pain and tenderness**: I. HYPERTROPHIC CIRRHOSIS; II. FATTY LIVER; III. LARDACEOUS LIVER; and IV. HYDATID and other cysts. In CATARRHIAL JAUNDICE (§ 241), CHRONIC CHOLELITHIASIS,

<sup>1</sup> Rolleston and Turner, *The Lancet*, Dec. 16, 1899.



and some other disorders, the liver is somewhat enlarged, but this is not their main feature.

1. *The liver is enlarged and PAINLESS ; its surface is hard, JAUNDICE IS PRESENT, but little or no ascites, and there is a long history of failing health*—the disease is probably HYPERTROPHIC ALCOHOLIC CIRRHOSIS.

§ 253. **Hypertrophic Cirrhosis of the Liver** is a term employed in a generic or clinical sense to indicate a progressive enlargement of the liver, due to an increase in the connective tissue of the organ with a tendency to jaundice. The condition may occur under at least five different aspects, due respectively to Alcoholism, Syphilis, Gall-stones, Chronic Heart-disease, and Malaria. Biliary cirrhosis may also be associated with Splenic Anæmia. A rare variety of hypertrophic cirrhosis accompanied by pigmentation of the skin or bronzing, has been described. Sometimes this is attended by glycosuria, and has been called "BRONZED DIABETES." The pigmentation resembles that of Addison's disease, but the liver is larger than in that disease. The pigment contains iron.<sup>1</sup>

Ia. HYPERTROPHIC ALCOHOLIC CIRRHOSIS (syn. biliary cirrhosis). In this form of cirrhosis the fibrous overgrowth occurs around single lobules, hence the name "uni-lobular cirrhosis," applied to this form. The disease is now generally recognised as a distinct form of alcoholic liver. The organ is enlarged *throughout the whole course* of the disease ; there is a great tendency to jaundice, and but little ascites—features which contrast with those of the more common condition, Atrophic Alcoholic Cirrhosis, just described.

*Symptoms.* 1. The symptoms come on very insidiously, with a failure of the general health. The patient rarely applies for medical aid until (2) jaundice has set in, which may be very pronounced. 3. Fever in some cases occurs at intervals, and may be as high as 103°. 4. In spite of the intense jaundice there are few or no signs of portal obstruction, and ascites is rarely if ever present. 5. The liver is uniformly, and may be considerably enlarged, hard, and sometimes rough. There is no tenderness

<sup>1</sup> Osler, *Lancet*, August 12, 1899, p. 440 ; *Lancet*, August 26, 1899, and *B. M. J.*, vol. ii., 1899, p. 1595.

and no actual pain (except during the feverish attacks), though a dull weight may be complained of in the hepatic region. The spleen is normal or only slightly enlarged. 6. A history of alcoholism is usually present, but in many cases the cause is obscure.

*Diagnosis.* In *atrophic alcoholic cirrhosis*, after some months of preliminary enlargement the liver undergoes contraction (§ 252). The percussion area of the liver, except in the early stages, is therefore normal or diminished. There is less tendency to jaundice, but ascites occurs in most of the cases. *Fatty* and *amyloid* livers are not accompanied by jaundice. *Cancer* has a more rapid and painful course. And see Table XXI.

*Prognosis.* Sometimes patients die within twelve months, with an acute onset of the typhoid state, but most live for a number of years, with signs of progressive emaciation. The prognosis is more favourable than in the atrophic form of cirrhosis; recovery is fairly common.

The *treatment* of Congestion (§ 240), or Atrophic Cirrhosis, is applicable, according to the predominating symptoms.

**IB. CARDIAC VALVULAR DISEASE** results as we have seen in very considerable congestion of the liver. Long-continued passive engorgement of the liver gives rise to changes known as the "nutmeg liver," and this may be attended by a considerable degree of fibrosis, accompanied by more or less enlargement of the organ. The diagnosis depends on the presence of C.V.D. and other features (see Passive Congestion, § 257).

**IC. SYPHILITIC DISEASE** of the liver generally takes the form of a diffuse hypertrophic fibrosis; or it may be met with in the form of *gum-mata*. Undoubtedly hepatic fibrosis may result from both hereditary and acquired<sup>1</sup> syphilis, though probably the gummatous form is commoner in the latter. In the inherited variety, bands of fibrous tissue pass through the liver in various directions, causing sometimes great distortion.

The *symptoms* are somewhat variable. The liver is moderately enlarged; there is not much tendency to jaundice and portal obstruction excepting in the final stages. There may be actual pain, especially when the capsule of the liver is involved; but as a rule there are only indefinite sensations of illness, accompanied in the gummatous cases by a slight degree of intermittent pyrexia. In the gummatous form nodular projections may possibly be made out on the surface of the organ. The presence of such projections.

<sup>1</sup> I have met with several cases of marked diffuse fibrosis of the liver due to acquired syphilis. Glisson's capsule was extremely thick, and large ramifying bands passed from it into the organ in all directions.

accompanied by intermitting fever and a history of syphilis, in a young adult practically make the diagnosis certain.<sup>1</sup> In the absence of a syphilitic history the occurrence of pain and local tenderness at intervals points to syphilitic rather than to alcoholic cirrhosis, because *perihepatitis and the involvement of the capsule* are prominent features of syphilitic cirrhosis.<sup>2</sup> In the diagnosis from cancer we have mainly to rely on the efficacy of iodide and the (usual) absence of jaundice and ascites in syphilitic disease.

The *prognosis* as a rule is good, if the nature of the disease be discovered and it is treated by large enough doses of potassium iodide and other antisymphilitic remedies.

**Id. CIRRHOSIS OF BILIARY OBSTRUCTION.** Hypertrophic cirrhosis has been produced experimentally in one half of the liver by ligature of one hepatic duct, and some observers have met with it clinically in association with gall-stones. When acting as clinical clerk to the late Dr. Charles Murchison I had the opportunity of observing a case of this kind which was under his care. The patient was a woman, aged 45, with a history of repeated attacks of biliary colic. There was great enlargement of the liver, with jaundice of three years duration. Cancer was excluded from the diagnosis, as the patient lived for some years afterwards.. Hypertrophic cirrhosis due to biliary obstruction was Dr. Murchison's diagnosis, or it may have been due to stenosis of the hepatic duct.

**Ie. MALARIAL CIRRHOSIS.** Subjects of prolonged malarial poisoning have an enlarged liver, which is believed to be due to cirrhosis. Alcohol may possibly be a contributory cause in these cases.

**II. The enlargement of the liver** is PAINLESS and uniform; the surface is smooth and soft; there is NO JAUNDICE OR ASCITES, and the SPLEEN IS NOT ENLARGED; there is a history of alcoholism, or the patient is suffering from *phthisis*—the disease is probably FATTY LIVER.

§ 254. **Fatty Liver** is a condition in which fat is deposited in the hepatic cells, commencing in the periphery of the lobules. It is nearly always associated with some other disease.

*Symptoms.* (1) The liver is enlarged uniformly and is quite smooth. (2) Pain, jaundice, and portal obstruction are absent. (3) The accompanying symptoms are due to the cause of the fatty liver, and may consist therefore of debility, anæmia, etc. (4) The history of a *cause* is important, viz., (i.) Chronic wasting disease, such as *phthisis*. (ii.) Fatty liver appears in association with fatty heart (*q.v.*) and general obesity. (iii.) It often occurs consequent on chronic alcoholism; and a mixed degeneration of fat and fibrosis is not uncommon.

<sup>1</sup> A case of this kind is recorded by Bristowe in the Clin. Soc. Trans., xix., p. 240.

<sup>2</sup> Cheudle, Lumleian Lectures, B. M. J., p. 766, vol. i., 1900.

The *diagnosis* from the painful enlargements of the liver is not difficult (see Table XXI.). From *lardaceous* liver it is known by the absence of signs of lardaceous spleen or kidney, and the presence of their respective causes.

The *prognosis* and *treatment* depend upon the primary disease, *i.e.*, the cause. It is hardly likely that the fat can be removed.

III. *The enlargement of the liver is* UNIFORM and PAINLESS ; *the surface is smooth and hard ; there is* NO JAUNDICE, NO ASCITES ; *the* SPLEEN IS ENLARGED ; *there is a history of prolonged purulent discharge or constitutional syphilis*—the disease is LARDACEOUS DEGENERATION.

§ 255. **Lardaceous (amyloid or waxy) Liver** is a condition of amyloid change starting in the capillaries and smaller arteries of the organ, leading sometimes to an immense enlargement.

*Symptoms.* (1) The liver is enlarged uniformly and smoothly, and feels firm and resisting to the palpating hand. (2) Pain, jaundice, and portal obstruction are absent. (3) The constitutional symptoms are due to the presence of the causal condition, and to the presence of amyloid disease of other organs.

*Etiology.* (i.) Long suppuration and purulent discharge, as from necrosed bone ; (ii.) constitutional syphilis ; and (iii.) tubercular disease of the lungs or elsewhere may produce amyloid disease here as elsewhere.

*Diagnosis.* The presence or history of a cause renders the diagnosis of amyloid disease comparatively easy. See also Table XXI.

The *prognosis* depends upon the amount of amyloid disease elsewhere. Diarrhœa indicating amyloid changes in the intestines, abundant pale urine with albuminuria indicating amyloid disease of the kidneys, are untoward signs. If the cause is remediable, as by surgical treatment, the liver may decrease in size.

*Treatment.* The indications are (i.) to remove the cause, and (ii.) to keep up the strength. The former is attained by administering pot. iod. in the case of syphilis, though only with partial efficacy, and by surgical treatment in the case of long-standing discharges. Tonics such as iron and quinine with cod liver oil are useful.



IV. The enlargement of the liver is PAINLESS, but NOT UNIFORM, and the upper margin of the liver dulness is perhaps *ARCHED*; there is no jaundice or ascites, and the spleen is not enlarged; a thrill or vibration is felt on percussion—the disease is *HYDATID CYST*.

§ 256. Hydatid tumour of the liver depends on the presence in the liver of a parasite rare in this country, though common in Australia, India, and Iceland, where dogs live in close association with man; and in Russia, where wolves are common.

*Symptoms.* (i.) There is a slowly increasing enlargement of the liver, which is smooth, globular, and elastic, sometimes fluctuating. The right chest may be bulged outwards with dulness in the axilla. When the fingers of the left hand are laid on the tumour and tapped with those of the right hand the “hydatid fremitus,” or “vibration,” is felt in some cases. This is a peculiar thrill thrown back upon the hand by a large quantity of limpid fluid. (ii.) Pain is absent unless the tumour is very near the surface, when great pain may be present, because the capsule is involved. (iii.) No constitutional symptoms appear unless the tumour becomes so large as to press upon the surrounding structures, or becomes inflamed and suppurates.

*Etiology.* The parasite enters the alimentary canal of man by means of drinking water contaminated by the fæces of the dog or wolf, containing the ova of the *tænia echinococcus*, i.e., tape-worm of the dog. The embryo is carried to the liver, where it encysts and grows. The cyst so developed has a firm gelatinous wall, and contains a clear fluid: and from the wall a number of proscolices or embryonic heads develop, each with a crown of most characteristic hooklets.

*Diagnosis.* Abscess of the liver has pain and fever, and on aspiration yields grumous material like anchovy sauce. Pleuritic effusion on the r. side, leading to dulness in the axilla, may resemble hydatid; in such cases a bulging outwards of the lower ribs over the liver points to the presence of hydatid. A renal cyst has resonance in front, due to the presence of the colon. A history of residence in Australia, etc., should lead one to suspect hydatid in cases of slowly increasing enlargement of the liver with few other symptoms. Exploratory puncture settles the diagnosis. This must be performed by a long trochar and cannula pushed in many directions—some 7 at least. The fluid withdrawn is pathognomonic. It is clear, opalescent, of low specific gravity, and contains large excess of chlorides, no albumen (unless inflammation has taken place), and—most characteristic of all—echinococcus hooklets (see Fig. 75).

*Prognosis.* The patient may live for several years with no other symptom than a slow increase in the size of the liver. Occasionally the tumour undergoes spontaneous cure by drying up. In some cases hydatids



Fig. 75.—Hooklets from an *HYDATID CYST* in man; the *TÆNIA ECHINOCOCCUS* (the tape-worm of the dog). Magnified about 150 times. These form the crown of hooklets around the anterior end of the scolex, and are absolutely distinctive of hydatid fluid. From a photomicrograph by Mr. Fredk. Clark.

appear in other parts of the body, as in the lung, peritoneal cavity, brain, or other organs. Jaundice may very occasionally be caused by cysts lodging in the bile ducts. The cyst may suppurate, giving rise to the symptoms of liver abscess, or pyæmia may be set up. Sometimes death occurs by the sudden rupture of the cyst into the pleura or peritonæum.

*Treatment.* Success has been attained in some cases by drawing off a few ounces of the fluid—the cyst then having shrivelled up. Otherwise free incision is the best method of treatment.

There are three diseases in which **enlargement of the liver** is attended **with pain and tenderness**: I. CHRONIC CONGESTION, II. CANCER OF THE LIVER, and III. ABSCESS OF THE LIVER. In CHRONIC CHOLELITHIASIS and several ACUTE DISORDERS the liver may be slightly enlarged and tender.

I. *The enlargement is moderate, smooth, and uniform, PAINFUL, and TENDER; some jaundice and ascites may be present, the SPLEEN IS ENLARGED, and there are signs of congestion of the abdominal viscera*—the disease is probably CHRONIC CONGESTION OF THE LIVER.

§ 257. **Chronic Congestion** of the liver is a condition in which the enlargement is due to venous obstruction (passive congestion). Compare the opening remarks in § 240.

*Symptoms.* (i.) The liver is tender, and a sensation of weight and fulness is complained of in the hepatic region. Pulsation may be conveyed to the palpating hand synchronous with the heart in the early stages, but as the organ becomes firmer this is lost. (ii.) Signs of general venous obstruction appear. (iii.) Ascites develops, and the spleen is enlarged. The yellow discoloration of jaundice may arise. (iv.) Gastro-intestinal disturbances are common.

*Etiology.* Passive congestion is the result of any backward pressure due to obstruction of the circulation. In most cases this is caused by heart or lung disease, and especially mitral valvular disease. Any growth pressing on the inferior vena cava above the diaphragm has similar effects.

The *diagnosis* is often aided by the recognition of the heart disease on which it depends. In some cases of *ascites* with anasarca of the legs, we may find both *hepatic enlargement* and *albuminuria*, and a difficulty may arise as to which was the primary cause of the condition—heart, liver, or renal disease. This difficulty is still further increased if extensive bronchitis prevents accurate auscul-

tation of the heart. Now, in such cases, *the liver* may be excluded as the primary cause, if the dropsy in the legs clearly preceded the dropsy of the abdomen. The presence of hepatic enlargement is then a sign of great value as helping to exclude *renal* mischief, because enlargement of the liver is not a usual sequence of kidney disease, although it is a fairly constant result of *cardiac* valvular disease.

*Prognosis.* The prognosis is altogether influenced by the cause of the congestion; and the state of the heart is generally the measure upon which the patient's chance of a longer or shorter life depends. In mitral stenosis an enlarged liver with ascites is less grave than in mitral regurgitation, because it normally occurs at an earlier stage in stenosis (Broadbent). It is most serious in aortic disease, and especially regurgitation.

The *Treatment* is that of the cause, and our attention must be directed to the heart and lungs. Purgatives and light foods are necessary in order to relieve the strain on the portal system. Leeches over the liver or bleeding may be indicated. (See also *Acute Congestion*, § 240.)

II. *The enlargement of the liver is* IRREGULAR; *the PAIN and tenderness may be great; JAUNDICE and ASCITES are present; the spleen is not enlarged; the patient is advanced in years, feeble and emaciated*—the disease is **CANCER OF THE LIVER.**

§ 258. **Cancer** of the liver may be primary, but is usually secondary to disease elsewhere; generally the stomach, rectum, or other part of the alimentary canal.

*Symptoms.* (i.) Pain is an almost constant feature of cancer of the liver; it is continuous, with exacerbations, and is independent of food or position. A certain amount of tenderness develops. (ii.) The enlargement of the liver is irregular, and nodules may be made out.<sup>1</sup> These are of a hard consistence, and increase rapidly. (iii.) Jaundice is usually present, *sooner or later*, and is intense and progressive. An intense jaundice persisting over 5 to 7 weeks in an old person should indeed always lead one to suspect cancer. Ascites generally occurs, either from involvement

<sup>1</sup> There is also a less common diffuse form of cancer in which there are no nodules, and in which the liver is only slightly and uniformly enlarged. In the nodular form the liver may be enormously enlarged.

of the glands in the fissure, or the peritoneum.<sup>1</sup> The spleen is not enlarged. (iv.) The general health of the patient is bad, and emaciation and cachexia may be present before any local signs are discovered. Cancer may be present in another part of the body. Fever occurs at intervals, especially in cases of primary cancer.

*Causes.* Cancer occurs after middle life; it is rare before 35. It is liable to occur secondarily to cancer of the stomach or rectum. When a patient has been troubled with gall-stones for a long period of time cancer of the liver is apt to result in later life.

*Diagnosis.* Jaundice is very rarely entirely absent in cases of cancer. This and the cachexia alone may justify a diagnosis of the condition. The diagnosis from *cirrhosis* may be difficult when nodular enlargement cannot be definitely made out, and when considerable ascites is present. In cirrhosis there is little or no pain and tenderness, the history of the illness is of longer duration, the spleen is enlarged, and the jaundice is not so intense. The *inflammatory thickening* under the liver after a long history of gall-stones may resemble cancer, and can be distinguished only when time shows little or no increase in size of the enlargement. *Syphilitic* liver has not so much pain and tenderness, is of slower growth, and very rarely produces ascites.

*Prognosis.* Cancer of the liver is usually fatal within six to twelve months, death taking place from exhaustion. Untoward symptoms are rapid growth, ascites, or respiratory difficulties due to extension of the disease to the lungs.

*Treatment* can be palliative only. Morphia or opium is administered for the pain, and attention must be given to the relief of the symptoms of gastric distress, and to aid nutrition. With rest and care the patient may have periods during which the disease makes no progress, and which hold out to the patient false hopes of his ultimate recovery.

III. **Abscess of the Liver** also produces considerable hepatic enlargement which is PAINFUL and TENDER. It has already been described among the Acute Diseases, § 246; but sometimes it runs a very chronic course.

<sup>1</sup> Dr. Chas. Murchison used to teach that *jaundice* with *ascites* in an *old person* usually indicated cancer.



**Tumours of the Liver**, other than CANCER (§ 258, *ante*), HYDATID (§ 256), and GUMMA (§ 253, *Ic*) are more rare. Their presence is manifested by *enlargement of the organ*, which may be regular or irregular, accompanied in some cases by constitutional symptoms. When, as in some cases of ACTINOMYCOSIS (§ 218), they assume an inflammatory form, there is, of course, pyrexia, accompanied mostly by sweatings and rigors. SARCOMA OF THE LIVER is occasionally met with, *e.g.*, Lympho-sarcoma, but it is most often secondary to deposits elsewhere, and the liver condition is only a subordinate part of the case. The patient may be younger than in the other form of malignant disease. Chondro-sarcoma, Melano-sarcoma, Tubercle, and Fibroma are met with on very rare occasions.

§ 259. **Floating Liver** (Dropping or Ptoxis of the Liver, Hepatoptosis) is probably a somewhat rare condition<sup>1</sup> which is due to a laxity of the ligaments. It has lately been receiving some attention (*vide The Lancet*, May 12, 1900). It is apt to be mistaken for enlargement of the organ, and *vice versa*. The condition has been referred to under Abdominal Pain (§ 175), because, if attended by symptoms, this is the principal one. There may also be vague neurasthenic symptoms.

## THE SPLEEN.

There is still some doubt as to the precise part which the spleen plays in the economy; and symptoms may be altogether wanting when it is diseased. Great diminution in size of the organ has been found *post-mortem* without any symptoms during life; and the spleen has been removed by operation without a fatal issue.<sup>2</sup> Sometimes, however, profound disturbance attends enlargement of the organ.

### PART A. SYMPTOMATOLOGY.

§ 260. In addition to the local pain and discomfort due to the enlargement of the organ, the symptoms which arise may include extreme pallor of the skin (the most intense anæmic pallor known is that associated with splenic leukæmia), great weakness, and enormous increase in the number of leucocytes in the blood; but we are not sure that all of these are results of splenic disease. In other instances, in "ague cake" for example, very great enlargement takes place without any symptom beyond the inconvenience due to the size of the organ. The *symptom* which is found to be most constantly associated with disease of the

<sup>1</sup> Some dispute its rarity, *e.g.*, Glénard, *loc. cit.*

<sup>2</sup> Spencer Wells has reported several cases of removal of the spleen, and in each instance the patient seemed in good health for at any rate some years after the operation. More recently Mr. Bland Sutton has reported 9 cases of extirpation of the spleen for various maladies (Report Clin. Soc., Lond., *The Lancet*, 1900, vol. ii.).

spleen is anæmia, the various "causes" of which will be discussed elsewhere (Chapter XVI.).

PART B. PHYSICAL EXAMINATION.

§ 261. The only physical signs which can be relied upon as diagnostic of splenic disease are the signs of enlargement of the organ, and this is most readily made out by **palpation**. When the spleen is enlarged, the anterior edge of the organ being free makes its way downwards and forwards towards the umbilicus. The *notch* in the anterior border is so characteristic that it forms a strong point in diagnosis of any splenic tumour. **METHOD.** Stand on the right side of the patient, who should be lying on his back. Pass the left hand across the abdomen, and lay it posteriorly over the eleventh rib on the left side, and place the right hand flat upon the anterior surface of the abdomen, with the tips of the fingers just below the eleventh rib. By gently dipping them down into the abdomen, and tilting the organ upwards with the left hand during inspiration, the splenic notch may be felt if the organ is enlarged. It is more readily palpated when the patient draws a deep breath. Normally, the spleen cannot be detected by palpation, and even slight enlargements may not always be appreciated. An enlarged spleen always has a space between its posterior edge and the erector spinæ behind, into which the fingers can be dipped—at any rate in spare subjects. *Fallacies.* Without being enlarged, the spleen is readily palpable when it is displaced downwards, or is "floating." It is sometimes displaced downwards in cases of deformed chest (*e.g.*, rickets), large pleuritic effusions, and emphysema.

§ 262. The **percussion** of the spleen is attended with some difficulty. The organ is situated in the left hypochondrium, between the upper border of the ninth rib and the lower border of the eleventh; and roughly between the mid-axillary and scapular lines (Fig. 39, p. 165). It extends obliquely forwards and downwards nearly to the costal margin. It lies wholly beneath the ribs, and the upper  $\frac{1}{3}$  is overlapped by the lung. Percussion does not afford a very accurate means of investigation, but it is well to remember that *a straight line drawn from the centre of the l. axilla, obliquely downwards and forwards to the umbilicus,*

should be resonant in its entire length (Gairdner's line). The spleen normally lies altogether behind this line, but if it be enlarged this line is impinged upon, by dulness at the junction of its middle and lower thirds.

The SURFACE LANDMARKS of the spleen may be said to form an oval, lying *obliquely* between the post-axillary and mid-axillary lines, and having, for purposes of description, four borders. The procedure for percussing out the *anterior* and *lower* borders differs from that used to elicit the *upper* and *posterior*, because the latter recede from the surface, the lung intervening. It is best to percuss at the end of an expiration, because the spleen is then less covered by lung. As mentioned above, palpation is preferable, but to define the *anterior* and *lower* limits by percussion the patient should lie on his back. 1. *Anterior border*—percuss *lightly* along the tenth rib, starting at its anterior end, and the note will be found to become dull about the *mid-axillary* line. 2. For the *lower border* percuss, also *lightly*, along the posterior-axillary line from below upwards, and the lower border should be reached about the lower edge of the eleventh rib. 3. To define the *upper* and *posterior* limits is very much more difficult, and very often, in fat subjects for instance, it is impossible. Fortunately it is not of so much importance to map out the posterior border. The patient must either sit up or lie in a semi-prone position, resting on his r. scapula. If he turns completely on to his r. side the spleen may fall away from the l. side. His l. hand should be placed on his head. *Upper border*—percuss with a *heavy* stroke just behind the post-axillary line, starting from the angle of the scapula and working vertically downwards. After repeating this several times, it will be noticed that the pulmonary resonance is impaired at the upper border of the ninth rib. 4. *Posterior border*—similarly with heavy percussion, by starting over the neck of the tenth rib and continuing along that rib anteriorly, you may elicit a change of note just in front of the scapular line.

1. *Fallacies.* The dulness of *splenic enlargement* may be simulated by pleuritic effusion or consolidation of the l. lung. The area of splenic dulness may be *diminished* by emphysema of the lungs, or distension of the stomach or the colon by gas. The splenic dulness may be altogether absent when there is a wandering spleen, or congenital absence of the organ.

#### PART C. DISEASES OF THE SPLEEN.

§ 263. The diseases of the spleen are all—if we except the relatively rare cases of wandering spleen and atrophy—comprised under the causes of **enlargement of the organ**, and its **diagnosis** therefore becomes a matter of considerable importance. Enlargement is detected by palpation aided by percussion as above mentioned. The mechanical effects of pressure, when the spleen is very much enlarged, are mainly dyspnoea and gastro-intestinal disturbance.

SPLENIC ENLARGEMENTS have three chief characteristics :— (1) The splenic *notch* is felt on its anterior border ; (2) the mass moves with respiration ; (3) it is dull to percussion because the resonant colon does not lie in front of splenic tumours, as it does in front of renal tumours, Gairdner's line of percussion resonance (*vide supra*) being thus impinged upon. (4) It is also worth bearing in mind the fact that when an area of dullness is due to splenic enlargement, its outline *resembles in shape* that of the normal spleen. A splenic enlargement or tumour may further be diagnosed from other abdominal tumours by remembering (5) its mobility on bi-manual palpation, unless bound down by adhesions ; and (6) its surface, which in simple enlargement of the organ is distinguished from neoplasms of the peritoneum, stomach, intestines, etc., by being smooth and firm.

Splenic enlargements or tumours may have to be diagnosed from the following conditions :—(1) *Renal tumours*, and especially movable kidney, in which (i.) there is resonant intestine in front of the tumour, and (ii.) there is not, as in splenic tumours, any resonant percussion in the flank ; (2) *Enlargement of the l. lobe of the liver*, in which (i.) the dullness is continuous with that of the r. lobe, whereas splenic dullness rarely reaches to the middle line, and (ii.) the splenic notch is absent ; (3) *Cancer of the cardiac end of the stomach*, in which (i.) the dullness is less absolute, (ii.) there is no notch, and (iii.) there is coffee ground vomiting, etc. ; (4) *Ovarian tumour*, which (i.) will have grown from below upwards, (ii.) the hand cannot be pushed between the tumour and the pelvic brim as it can in the case of a splenic tumour, and (iii.) can be felt on vaginal examination ; (5) *Accumulation of feces*, in which (i.) the tumour has an irregular outline, (ii.) doughy consistence, and (iii.) a course of purgatives and enemata will remove it ; (6) *Post-peritoneal tumour*, in which (i.) there is no notch, and (ii.) no resonance behind it ; (7) *Abdominal aneurysm*, which, when of sufficient size to be mistaken for the spleen, is attended by (i.) severe pain in the back, and (ii.) evident expansile pulsation ; (8) *Deep-seated abscess in the abdominal parietes*, in which the swelling is (i.) tender and has a vague irregular outline, and (ii.) situated more superficially than a splenic tumour.



The causes of enlargement of the spleen may be grouped as follows :—

a. **Acute enlargement.**

I. Poisons in the blood.

II. Embolism of the spleen.

b. **Chronic enlargement.**

I. Cardiac disease.

II. Hepatic disease.

III. Lardaceous disease.

IV. Splenic Leukæmia.

V. Hodgkin's disease.

VI. "Ague cake."

VII. New growths—Hydatid, cancer, tubercle.

VIII. Constitutional syphilis.

IX. Rickets.

X. Splenic anæmia.

If the illness of which the patient complains came on gradually and is running a **chronic** course, turn to § 265; but if it came on more or less suddenly, commence at **acute diseases**, § 264.

§ 264. **Causes of acute enlargement** of the spleen, *i.e.*, coming on rapidly with acute symptoms, and often preceded and accompanied by pain or **tenderness**.

I. **POISONS IN THE BLOOD.** The spleen is enlarged more or less in nearly all the acute specific fevers, especially typhoid, typhus, relapsing fever, pyæmia, malarial affections, and pneumonia. This form is recognised by—(i.) the presence of symptoms of the primary malady; (ii.) the presence of pyrexia; (iii.) the subsidence of the enlargement with improvement in the primary disease.

II. **SPLENIC EMBOLISM** may be either *benign* or *septic* in character.

(a) If *benign* it follows as a complication of cardiac valvular disease, especially mitral stenosis; and is recognised by—(i.) the enlargement of the spleen rarely being great, and soon passing away; (ii.) a history of sudden onset of *acute pain* in the left hypochondrium, which rapidly subsides; (iii.) the absence of hepatic or pulmonary disease, and the presence of symptoms of cardiac valvular disease.

(b) If of *septic* origin it follows as a complication of malignant endocarditis (§ 42) or some other septicæmic process. Here inflammation of the splenic substance may proceed to the formation of an abscess. Septic embolism is characterised by the same symptoms as simple embolism, and in addition we have evidences of the pyæmic process, and symptoms of septic emboli in other parts.

§ 265. *The causes of chronic enlargement of the spleen are more numerous than those of acute. In adults the commonest perhaps is CONGESTION, due to chronic LIVER or HEART disease; but in children SYPHILIS and RICKETS are more frequent. In old age CANCER must be suspected, and in phthisical subjects LARDACEOUS DISEASE, or LEUKÆMIA.*

I. In CARDIAC DISEASE the spleen shares in the general congestion of organs in the later stages, but it is a relatively unimportant feature, and the enlargement is rarely great in uncomplicated cases.

II. HEPATIC DISEASE and other causes of PORTAL CONGESTION are attended by splenic congestion, and may result also in a fibrosis of the organ; but here again the splenic condition is only one of the aids to the diagnosis of the primary condition.

III. LARDACEOUS or WAXY DISEASE is one of the commonest causes of splenic enlargement. The swelling may be considerable, but it is rarely as large as V. or VI. below. It is recognised by—(i.) the surface being smooth and uniform; (ii.) painless enlargement (waxy disease) in other organs, such as the liver; (iii.) profuse diarrhœa, due to waxy disease of the intestines; (iv.) the history of a cause, viz., prolonged purulent discharge (*e.g.*, chronic phthisis), or of syphilis.

IV. In SPLENIC LEUKÆMIA the spleen may be very large, extending even to the pelvis, and weighing over 10 lbs.; (i.) there is great pallor and debility; (ii.) leucocytosis is present, with changes in the corpuscles (see Leukæmia, Chapter XVI.); (iii.) there is great tendency to hæmorrhage from the nose and other mucous membranes; (iv.) frequently diarrhœa is present, and, if the disease lasts for long, ascites may supervene.

V. In HODGKIN'S DISEASE the spleen is occasionally the seat of lymphadenomatous enlargement. It is recognised by—(i.) the enlargement being slight; (ii.) the presence of enlarged lymphatic glands in other parts of the body; and (iii.) the paroxysms of pyrexia and glandular swelling.

VI. "AGUE CAKE" is a term used to indicate the persistent enlargement and induration of the spleen due to malaria. (i.) The organ is smooth and hard, and it may be of very great size, although causing comparatively little inconvenience. (ii.) There is a history of ague, or of exposure to malaria, and great debility and anemia

are usually present. (iii.) There is an absence of heart or liver disease. (iv.) There is little or no increase in the number of white corpuscles, and no other signs of leukæmia are present.

VII. NEW GROWTHS of the spleen are recognised chiefly by the irregularity of the enlargement to which they give rise.

1. HYDATID of the spleen often exists concurrently with hydatid of the liver, and is recognised by—(i.) the enlargement not being uniform; (ii.) the evidences of a fluid cyst, fluctuation, and the “hydatid thrill”; (iii.) an exploratory puncture draws off clear, opalescent, non-albuminous fluid of low specific gravity (§ 256).

2. CANCER of the spleen is extremely rare as a primary, and comparatively rare as a secondary deposit. It is known by—(i.) the hardness and irregularity of the tumour; (ii.) the severe pain; (iii.) cachexia, and cancer elsewhere.

3. TUBERCLE sometimes affects the capsule of the spleen, but the enlargement is not great, and it is not of much clinical importance, as there is generally more advanced tubercle in other organs.

ENLARGEMENT OF THE SPLEEN IN CHILDREN is a fairly common occurrence, and it may be due to constitutional SYPHILIS, RICKETS, or SPLENIC ANÆMIA.

VIII. CONSTITUTIONAL or HEREDITARY SYPHILIS may produce three histological changes in the spleen—*fibrous infiltration*, or simple hypertrophy as it is sometimes termed, *lardaceous disease* (*vide supra*); and *gumma* (which are comparatively rare). SYPHILITIC FIBROUS INFILTRATION is differentiated by—(i.) it only occurs in children who are the subjects of hereditary syphilis; (ii.) it is usually accompanied by snuffles, rash, and other syphilitic manifestations (p. 33); (iii.) it subsides under anti-syphilitic treatment. GUMMA is sometimes met with under the same conditions; occasionally also in adults.

IX. RICKETS causes an enlargement of the spleen, and this form is recognised by—(i.) its occurrence in early childhood; (ii.) the accompanying swelling of the epiphyses, deformity of the chest and head; and (iii.) there is often enlargement of the liver as well.

X. In SPLENIC ANÆMIA the spleen is enlarged, but anæmia is the chief symptom. There is little or no leucocytosis, but the red corpuscles are diminished.

*The treatment and prognosis* of splenic enlargement depend, for the most part, on the primary condition. The treatment of lardaceous disease and of hydatid is given under hepatic disorders (§§ 255, 256). The treatment of "Ague Cake" consists of—(i.) removal to a non-malarious district, and the administration of quinine and tonics, with free saline purgation; (ii.) ung. hyd. iod. dil., rubbed over the splenic area, was a remedy advocated by Murchison. (iii.) Violent movement must be forbidden, as the spleen may rupture.

§ 266. **Wandering Spleen** (Floating, Dropped, or Dislocated Spleen, Splenoptosis) may readily be mistaken for enlargement of that organ when met with in the lesser degrees of displacement. But when the dislocation is, as generally happens, considerable, it is more often taken for a floating kidney. However, the presence of the notch, the fact that it can be made to recede upwards, and that it comes down in front of the colon, aid in the diagnosis. The condition is mostly met with in multiparae with pendulous abdomens. It may be accompanied by nervous symptoms, though less constantly than in dislocation of some of the other viscera. If troublesome, the condition may be relieved by removal of the organ, an operation which has been performed several times with good results (Bland Sutton, see footnote <sup>2</sup>, p. 461).

§ 267. **Atrophy of the Spleen** is as a rule unattended by any symptoms. It is, as Bristowe says, a condition not infrequently met with. It may be congenital; but its commonest causes are—  
I. CIRRHOSIS of the spleen, due to an increase in the interstitial tissue, the result, as in cirrhosis of the liver, of alcohol; and  
II. CONTRACTION OF THE FIBROUS CAPSULE, usually of syphilitic origin. The syphilitic deposits in the capsule of the spleen sometimes take on a cartilaginous change, and form plates of cartilage. I have come upon them several times in the dead-house, but they had been unattended by symptoms during life. I have also met with some 5 cases of marked atrophic condition of the spleen, death having occurred from independent causes.



## CHAPTER XIII.

### THE URINE.

THE intricate subject of renal diseases is rendered more comprehensible by a brief consideration of their history. In 1812 Dr. Wells<sup>1</sup> found that albumen in the urine was associated with certain forms of dropsy. It was not however until 1836 that Bright<sup>2</sup> went a step further and discovered that dropsy and albuminuria when associated together (in the absence of heart disease) were indicative of disease of the kidneys. The term "Bright's disease" has thus come to be synonymous with disease of the kidney. We now know that there are many different disorders of the kidney which present dropsy and albuminuria at some stage of their course. Thus, we have *acute* inflammation of the renal epithelium, and this is known as "Acute Bright's Disease." Similarly, the term "Chronic Bright's Disease" should connote *chronic* renal disease, of which there are at least three recognised clinical varieties—chronic tubal nephritis, lardaceous disease, and chronic interstitial nephritis. But the term "Chronic Bright's disease" has, in later years, come to be used in a more limited sense, and is now more usually employed to indicate only the last-mentioned variety—chronic interstitial nephritis, *i.e.*, the granular or gouty kidney. In more recent times this form of renal degeneration has come to be regarded only as part of a widespread cardio-vascular change in which the general symptoms are far more pronounced than the urinary symptoms. In 1872 Drs. Gull and Sutton<sup>3</sup> made a study of the nature of these cardio-vascular changes and came to the conclusion that they consisted mainly of a widespread arterio-capillary *fibrosis*. Sir George Johnson<sup>4</sup> alone, at that time, disputed this. But now

<sup>1</sup> Wells, Transactions of the Society for the Improvement of Medical and Surgical Knowledge. Lond., 1812, iii. 194.

<sup>2</sup> Bright, Guy's Hosp. Rep., Lond., 1836, I.

<sup>3</sup> Trans. Roy. Med. Chir. Soc., 1872; *Lancet*, 1872.

<sup>4</sup> Trans. Roy. Med. Chir. Soc., Lond., 1873; *Brit. Med. Journ.*, 1872.

most observers are agreed that when a structural change takes place, it consists, at any rate in the earlier stages, of an increase in the muscular coat of the arterioles.<sup>1</sup>

Renal diseases are for the most part chronic, and often obscure; but, with a knowledge of these historical data, their study would be comparatively simple were it not for the confusion which has arisen largely owing to the numerous terms applied by pathologists to diseases which differ anatomically, though not always clinically. In what follows we shall be concerned only with the clinical aspect of renal disease.

It is not possible in actual practice to separate kidney diseases proper from disorders of other parts of the urinary tract, for changes in the urine are common to them all. It will be necessary, therefore, to refer to disorders of the bladder, prostate, and urethra for diagnostic purposes, though their treatment comes mainly within the province of the surgeon.

#### PART A. SYMPTOMATOLOGY.

The chief function of the kidneys is the elimination of nitrogenous waste. When this is interfered with by structural or functional disease a toxic condition results, which, when it reaches a certain stage, is known as uræmia.

As a consequence of the deep-seated position of these organs, the local symptoms referable to the kidney are, excepting in cases of Tumour or Displacement, of subordinate importance. The most constant and **CARDINAL SYMPTOM** of kidney disorders is some **Alteration in the Urine**, which as an indication of renal disease corresponds to the physical signs in other organs, and is dealt with in PART B. of this chapter. The cardinal symptoms next in order of importance are **Pallor of the Surface** and **Dropsy**. **General symptoms**, due to the toxic state which results from the retention of the nitrogenous waste, also accompany these diseases.

§ 268. **Pallor of the Surface and Malaise** are very constant

<sup>1</sup> Hamilton, "Text-book of Pathology"; Dickinson and Rolleston, *The Lancet*, July, 1895. The author's investigations into the large series of renal cases which presented themselves at the Paddington Infirmary also confirm this (*Trans. Med. Soc., Lond.*, 1897—1898; *The Lancet*, 1897, vol. i., pp. 882 and 1235; *Brit. Med. Journ.*, Jan. 23, 1897). With certain methods of preservation and hardening, or with insufficient staining, the middle coat presents precisely the appearance of fibrous tissue; but well-stained logwood preparations always reveal the rod-shaped nuclei, and acid orcein will always reveal the elastic tissue boundaries of the tunica media.

features of all organic kidney diseases. To the experienced eye the pallor differs from that of anæmia in a manner somewhat difficult to describe. The skin has a "waxy" hue, a simile which is still further exemplified when dropsy is present. It affects the whole body, but is always most evident in the face. In chronic interstitial nephritis (ch. Bright's disease) the pallor has a greyish hue. Other causes of pallor and their diagnosis will be given in Chapter XVI.

§ 269. **Renal dropsy** is of *general* distribution, in which respect it differs from cardiac dropsy, which starts in the *legs* or most dependent parts, and from hepatic dropsy, which starts in the *abdomen*. It is, however, most evident in the loose cellular tissue, *e.g.*, around the eyelids, where it is most marked on first rising in the morning. Towards evening the ankles become œdematous, or, as the patient may express it, a "ridge is present around the top of the boot." In severe cases (*e.g.*, in acute nephritis) the eyes may be almost closed by the swollen lids, and at the same time there may be signs of dropsy in the serous cavities—the pleura, peritoneum, and pericardium. Œdema of the solid organs also occurs in severe cases, and death may be produced by pulmonary œdema. Œdema glottidis is another serious though less frequent complication.

Dropsy is by no means an equally constant feature in all diseases of the kidney. In *acute and chronic parenchymatous nephritis* (*i.e.*, diseases in which the renal epithelium is primarily affected) dropsy is almost invariably present. But in *chronic interstitial nephritis* and *lardaceous kidney* it is comparatively rare; in the former of these two it may occur late in the course of the disease, when it is generally due either to cardiac failure or to secondary inflammation of the renal epithelium. In uncomplicated *pyelitis* and *neoplasms* dropsy is not present.

§ 270. A large number of **General Symptoms** occur consequent on the retention of the nitrogenous waste products—Cardio-vascular changes, hæmorrhages, breathlessness, affections of the nervous system, ocular changes, gastro-intestinal disorders, and secondary inflammations.

CARDIO-VASCULAR CHANGES frequently accompany renal disease. In acute and chronic renal disease there is mostly high arterial tension, and often dilatation of the heart. The accentuated second aortic sound which

accompanies this high tension is a useful indication in some of these cases for bleeding, or other measures for the reduction of arterial tension. In chronic interstitial nephritis the high tension is apt to be followed by a thickening of the arteries, due to hypertrophy of the muscular coat.<sup>1</sup> The left ventricle becomes hypertrophied and, in the later stages, signs of cardiac failure may ensue.

HÆMORRHAGES sometimes occur in chronic Bright's disease, a consequence of the high tension, combined in most cases with a diseased state of the blood-vessels. Epistaxis, for instance, may be the first symptom which leads to the discovery of chronic Bright's disease. Bleeding from the stomach and intestines, and purpura, sometimes occur. *Cerebral hæmorrhage is a frequent cause of death in this disease.*

BREATHLESSNESS, apart from that due to pulmonary œdema, is a common accompaniment of renal disease. A paroxysmal dyspnœa, coming on during the night in a person of advanced life, should lead us to suspect the existence of chronic Bright's disease, even although the patient may continue his occupation. *Cheyne-Stokes' respiration* may develop towards the end, with or without other symptoms of uræmia.

NERVOUS SYMPTOMS are not infrequent, apart from the cerebral hæmorrhage just referred to. Thus, *headache* is a symptom which accompanies all renal diseases, particularly those forms which terminate uræmia. Experience among the aged shows that in chronic interstitial nephritis is one of the most frequent causes of headache in advanced life. The patient may continue his work, and present no other symptom, but an examination of the urine may reveal the existence of chronic renal disease. *Vertigo, tinnitus*, and various *neuralgias* may also be complained of. *Insomnia* in the aged is another common symptom of chronic renal disease. The patient complains that he readily drops off to sleep, but as readily awakes, and that he may do so a dozen times every night. As the uræmic condition increases, however, *drowsiness* supervenes, which may pass into *coma*, with or without muttering *delirium*. Sometimes *convulsions* occur before death.

OCULAR CHANGES frequently accompany renal disease attended by albuminuria; and so characteristic are the changes that albuminuria may be diagnosed by their presence. Albuminuric retinitis comprises three alterations in both fundi—papillitis, flame-shaped hæmorrhages into the retina, and white spots of fatty degeneration.

GASTRO-INTESTINAL symptoms attend some renal diseases. Thus dyspepsia and irregularity of the bowels are common. *Vomiting*, when persistent, is a symptom of considerable gravity, because it is usually of toxic, *i.e.*, uræmic origin.

§ 271. The complications and secondary inflammations in renal cases are very apt to affect the SEROUS membranes, the MUCOUS membranes, and the SKIN—in a word, the limiting or “surface” structures of the body. The *serous membranes* often become inflamed insidiously, especially the pleura and pericardium. The effusion may sometimes come on very suddenly,

<sup>1</sup> As Dickinson and Rolleston have shown in *The Lancet*, July 20, 1895. See also Arterial Hypermyotrophy, § 71.



but the symptoms may be quite latent ; therefore the occurrence of severe dyspnoea in renal cases should lead us to suspect the sudden supervention of a serious pleural effusion (§ 22). In addition to the *pulmonary œdema* above mentioned, a low form of pneumonia and bronchitis are common complications of nephritis. Endocarditis is relatively rare. Within the last few years it has been recognised that various *skin affections* may supervene on renal disease apart from the fact that cellulitis is liable to affect dropsical limbs.<sup>1</sup> Amongst these may be mentioned eczema, urticaria, and various forms of erythema. Undoubtedly the most fatal is an epidemic form of exfoliative dermatitis, described by the author in 1891.<sup>2</sup> All the cases of renal disease complicated by the epidemic exfoliative dermatitis, which the author has since met with, have ended fatally.

§ 272. **Pain in the kidney.** Many serious diseases of the renal substance are unaccompanied by any pain or local symptoms. A sense of dull aching in the loins may be present at the onset of acute nephritis. In pyelitis, lumbar pain generally accompanies the appearance of pus in acid urine. The pain is extremely severe when the pyuria (pus in the urine) is due to a renal calculus (Renal Colic, § 262). Various tumours of the kidney are accompanied by pain, and perinephric abscesses are associated with lumbar pain and tenderness. A dull, dragging pain or weight in the lumbar region, relieved by rest in the recumbent posture, occurs with movable kidney ; it is usually on the affected side, and is liable to acute exacerbations resembling renal colic. The lumbar pain of renal disease must not be mistaken for the backache due to congestion of the female generative organs, nor for lumbago, in which the pain is usually of sudden onset, is not confined to one side, and may be accompanied by other rheumatic evidences. Less frequent causes of lumbar pain are aneurysm, cancer, and caries of the vertebræ.

§ 273. **Uræmia** is a term used to describe the collection of symptoms which arise from retention within the body of those nitrogenous constituents which are, under normal circumstances, elaborated into urea and eliminated by the kidney in the form of urea. The exact nature of these retained substances is not yet known. It used to be thought that it was urea which was retained (hence the name), but this is not so, though there is generally a diminution in the excretion of urea. The term uræmia is generally used for the intense *acutely* toxic condition which closes most renal cases ; but it may also be applied to the incipient or *chronic*

<sup>1</sup> Trans. Roy. Med. Chir. Soc. and Trans. Clin. Soc. Lond., 1899—1900.

<sup>2</sup> Trans. Med. Soc. Lond., 1891—1892 ; and *British Journal of Dermatology*, 1892.

condition which precedes this, and warns the observant physician of the gravity of the situation. They are the evidences of retention in the blood and the tissues of those substances which form a chain of compounds between the proteid food substances and the nitrogenous disintegration on the one hand, and the nitrogenous output (both quantitative and qualitative) on the other. Thus uræmia may arise in many hepatic diseases (defective elaboration, as mentioned in the introduction to Chapter XII.), and in many renal disorders (defective excretion).

Uræmia, more or less severe, may occur in almost all diseases of the kidney. In renal fibrosis (granular kidney) it occurs in a typically chronic form; in acute, subacute and chronic tubal nephritis it is the usual mode of death; in tuberculous, calculous and cystic disease, in hydronephrosis and consecutive nephritis, in active or passive congestion, and in lardaceous disease (rarely), mentioned in order of frequency, it is also apt to supervene. Moreover, without previous warning complete suppression of urine may produce death associated with symptoms of what is called *latent uræmia* (§ 313), in those relatively rare cases of blocking of the ureters.

*Symptoms.* Various types (nervous, gastro-intestinal, dyspnoëic, etc.) of uræmia are sometimes described; but it is more convenient and not more artificial to describe the symptoms under *incipient* and *advanced* chronic uræmia, and *acute* uræmia.

(1) In *incipient chronic* uræmia, such as occurs in a typical form in chronic interstitial nephritis, the symptoms are vague, and of gradual and almost imperceptible onset. The patient remains at work, but complains of malaise, loss of mental and bodily vigour, general wasting of muscular and subcutaneous tissues, impaired memory, and sometimes sleeplessness after the first few hours of the night. These, in addition to the urinary changes, may be the only indications of the condition.

(2) Symptoms of *advanced chronic* uræmia may succeed the foregoing, or may come on abruptly in a person apparently in perfectly good health. They consist of (i.) restlessness and muscular tremors (which are certainly one of the most constant symptoms); (ii.) persistent headache; (iii.) drowsiness during the day, with sleeplessness or "cat-sleeps" (dropping off for a few

minutes at a time) at night; (iv.) vomiting, without obvious dietetic irregularity or gastric disturbance, and sometimes diarrhœa; and (v.) dyspnœa on slight exertion (which is often the first symptom to be noticed), or coming on in paroxysms, especially at night.<sup>1</sup>

(3) *Acute* or *fulminating* uræmia may supervene at any stage of the foregoing, being ushered in perhaps by an increase of the headache, vomiting, or restlessness; or it may come on abruptly in an apparently healthy person. Its leading symptoms are three—(i.) low muttering delirium; (ii.) stupor, passing into coma, with or without (iii.) convulsions. In some cases of chronic Bright's disease convulsions or coma may constitute the first manifestation of the disease.<sup>2</sup> In some cases blindness (uræmic amaurosis), without appreciable ophthalmoscopic changes, follows the convulsions, and the condition may last for several days. In other cases deafness or local paralyses may ensue.

*Diagnosis.* Uræmia is known by the combination of the above symptoms, together with the presence of a cause, which can be made out on a careful examination of the urine. The diagnosis of uræmic coma is dealt with in Chapter XIX.

The *treatment* of uræmia is given under chronic Bright's disease (contracted granular kidney) (§ 298), in which malady both chronic and acute uræmia most typically occur.

#### PART B. PHYSICAL EXAMINATION.

**The examination of the urine** corresponds, in renal diseases, to the physical examination of other organs. It consists of (a) observing its *physical characters* (§ 274), viz., its appearance (*i.e.*, its colour, and whether it is clear or cloudy), its odour, reaction, specific gravity; the presence and characters of any deposit; and its diurnal quantity. (b) Then by *chemical analysis* (§ 279) we ascertain the presence or absence of albumen, the presence or absence of sugar, and other substances, according to circumstances. (c) Finally, a *microscopic examination* (§ 291) has to be made of

<sup>1</sup> Uræmic Dyspnœa may be: (i.) *Paroxysmal*; the attacks coming on chiefly at night, and resembling cardiac asthma (§ 21a). The patient sits up in bed gasping for breath, but there is no cyanosis, and the mind is clear. The breathing is often noisy, with a characteristic hissing quality (Addison). (ii.) *Continuous*, or continuous alternating with paroxysmal. (iii.) *Cheque-Stroke's Respiration* may last for weeks. The pulse slows in the apnoea period; and there is alternate contraction and dilatation of the pupil, the contraction occurring during the period of apnoea.

<sup>2</sup> This is explained by a sudden congestion of a chronically diseased kidney, and such cases (Coma or Convulsions occurring suddenly in an *apparently* healthy person) usually occurred during the winter in the Infirmary.

any deposit which may be present. It is important in all cases—not only in cases of suspected renal disease—to observe *and to note* the condition of the urine when the patient is first seen, even when the symptoms do not suggest renal disease.

### a. *Physical Characters of the Urine.*

§ 274. **Appearance.** The colour of the urine depends upon the proportion of pigments present. The chief pigments are urobilin and urochrome, whose antecedents are the blood and bile pigments; but there are many others.

The urine varies from a pale yellow to a deep amber, according to the DEGREE OF DILUTION of the pigments; and, as the latter are fairly constant in quantity, a *dark urine* is associated with a smaller diurnal quantity and a higher specific gravity than a pale urine. The urine is dark in excessive perspiration, acute nephritis, and pyrexial states generally. On the other hand, in certain diseases with *polyuria* the urine is *pale*, as in chronic Bright's disease, and in diabetes. In diabetes insipidus and hysteria the urine may be as colourless as water.

The colour of the urine may be altered by MORBID PRODUCTS, *e.g.*, a *dark orange colour to brown*, having a greenish tint on the surface with reflected light, is due to the presence of bile, and will vary in depth of tint according to the amount of bile present. A *red* colour, which may be a dark red or porter colour or only a mere "smokiness," is due to the presence of blood. In diseases in which there is destruction of the red blood corpuscles the urine is darkened, and this may be a means of distinguishing pernicious anemia from chlorosis. *Blackish-brown* colour may be due to melanin and certain oxyacids, and these cause the urine to darken on exposure. *Milky* urine is due to chyluria. Various DRUGS affect the colour of the urine. A *dark olive-green or black colour* may be due to the absorption of carbolic acid, as, for example, when this substance is used for dressings; or it may appear after the administration of such drugs as creasote, salol, tar, etc. The colour is explained by the presence of hydrochinon: which turns crimson on the addition of ferric chloride. A *reddish-brown colour* may be due to rhubarb, senna, or chrysophanic acid when taken internally, and a *bright yellow colour* follows the administration of santonin. All these turn red on the addition of an alkali. A *colourless* urine is said to result from tannin taken by the mouth, and a *reddish* hue from logwood.

**Urinary Deposit and Cloudiness** will be described in § 290.

§ 275. **Reaction.** The urine should be tested immediately or soon after being passed. In normal urine an acid reaction is found, turning blue litmus paper red, from the presence of acid phosphate of sodium. On standing for a time decomposition takes place, the urea being transformed into ammonium carbonate  $(\text{NH}_2)_2\text{CO} + 2\text{H}_2\text{O} = (\text{NH}_4)_2\text{CO}_3$ , and hence the reaction is alkaline. The same change takes place even within the bladder, in cases of chronic catarrh of that organ. Alkalinity due to a *fixed* alkali occurs even in normal urine after meals, or when a patient is undergoing alkaline treatment. A *neutral* reaction may occur under the same conditions. It is sometimes important to distinguish between the alkalinity due to a fixed alkali (*e.g.*, soda or potash salts), and that



due to decomposition, which depends upon a volatile alkali (ammonia). This is done by holding over a flame the red litmus paper which has been turned blue; if due to a volatile alkali, the red colour will return (as the volatile alkali is driven off). if to a fixed alkali, the blue colour remains.

§ 276. **Specific Gravity.** The average specific gravity of the urine varies between 1015—1025. It depends chiefly upon two substances normally present: urea and salts (especially chlorides); and the simple rule of doubling the last two figures, gives roughly a little less than the total quantity of solids present in parts per thousand. Extractives and pigment play only a small part; and practically—since the salts are fairly constant—the specific gravity, *in the absence of sugar*, gives us a fair measure of the urea present in a given sample. The specific gravity must be considered in relation to the quantity of urine passed; and to be able to draw accurate inferences from the specific gravity, the urine of a whole day should be collected, and a sample thereof tested (§ 278). The instrument used to test the sp. gr. is called a urinometer (Fig. 76). It is convenient to have a metal one with a flanged foot, as shown in that figure. The instrument must not touch the sides of the vessel, and the graduated stem should be read along the surface of the fluid, not at the place where it is raised along the stem by capillarity. These instruments are graduated for a temperature of 60° F. If the temperature of the urine is lower than this the true specific gravity is a trifle lower than the actual reading, and *vice versa*.<sup>1</sup>

§ 277. The normal odour of freshly passed urine is described as "aromatic"; it is very different from the ammoniacal odour of decomposing urine. The resinous portions of copaiba, cubebs, and other balsams are excreted by the urine, and impart their characteristic odour to it. Turpentine gives to urine an odour said to resemble violets. It may smell of volatile sulphides in presence of some microbes, notably *b. coli communis*.

§ 278. **The Diurnal quantity** varies considerably within the range of health. Normally, 40 to 50 ounces (1½ litres) are passed per diem, but the quantity depends upon the amount of fluid drunk, the action of the skin, and the activity of the renal circulation. In order to estimate the quantity of urea, and for some other purposes, it is necessary to collect the whole of the urine that is passed in 24 hours; say, for example, from 8 a.m. Monday, to 8 a.m. Tuesday. The patient should pass water at 8 a.m. on Monday morning, and this should be thrown away. Then all that is passed after that hour, together with what is passed at 8 a.m. on Tuesday, should



Fig. 76.—URINOMETER, made of metal, and with flanged foot (as supplied by Hicks, of Hatton Garden, E.C.). The flanges steady it while in the urine, and form a stand when not in use.

<sup>1</sup> When enough urine is not obtainable, and a glass bead urinometer is not accessible, mix the urine with one, two, or three times its own bulk of water and multiply the decimal figures of the specific gravity by two, three, or four respectively. For example, a mixture of one ounce of urine with three ounces of distilled water gives a specific gravity of 1005; the sp. gr. of the urine was 1020 ( $1005 \times 4 = 1020$ ).

be collected in one clean vessel, which must be carefully preserved from accident or interference. During the whole of that time it is necessary to pass water *before* going to stool, and to add this to the total collected. At 8 a.m. on Tuesday, after passing water and adding it to that previously passed, the whole should be stirred and measured. A specimen from this should then be put into a clean bottle (say 10 ozs.), and this should be labelled with the name of the patient, the date, and the total quantity passed in the 24 hours, and sent for examination immediately.

### b. Chemical Examination of the Urine.

Normally the urine consists of water containing about 4 per cent. of solids by weight, of which urea is the most important.

The following are the average quantities of the chief substances normally excreted in the urine in 24 hours:—

		Grammes.	Percentage.
Water	.	1,450 to 1,500	96.0
Solids	.	57 to 68	4.0
Consisting of:			
Organic.	Urea	28 to 32	2.5 to 3.0
	Uric Acid	0.7	0.05
	Hippuric Acid	0.3 to .2	0.15 to .10
	Kreatinine	1.7 to 2.0	0.10
	Pigments	Traces	
Inorganic.	Sodic Chloride	15 to 20	1.0 to 1.25
	Phosphates	2.5 to 3	0.16
	Sulphates	2.0 to 2.5	0.15
	Sodium	5.0 to 7	0.4
	Potassium	3.0 to 4	0.25
	Magnesium	0.4	0.03
	Calcium	0.3	0.02

In disease the three most important substances for which the urine has to be tested chemically are albumen, sugar, and urea.

§ 279. **Albumen** is the most frequent of the pathological constituents of the urine. The variety of albumen mostly present is serum albumen. (The other forms are given below.)

The chief tests for albumen are: (1) Cold nitric acid; (2) Boiling; (3) Picric acid.

1. *The Cold Nitric Acid test*<sup>1</sup> is the most delicate, accurate, and convenient test for small quantities of albumen in the urine. Pour some strong nitric acid into the bottom of the test tube, hold the tube in a very sloping position, and let the urine gently flow upon the top; a haze of precipitated albumen will appear at the line of junction. It is necessary to wait a few seconds for the haze to appear, when the albumen is very small in quantity; and the tube should be gently heated at the junction.

<sup>1</sup> If  $\text{HNO}_3$  is not handy, a saturated solution of common salt, carefully poured down the side of a test-tube containing albuminous urine gives a haze at the line of junction. But cold nitric acid was decided by a Committee of the Clinical Society of London to be the best all-round test for albuminous urine.

The *fallacies* of this test are not serious. (i.) Mucin, or urates, may form a precipitate, but it occurs *above* the line of junction ; (ii.) in a concentrated urine, a haze of tiny crystals of nitrate of urea may form, but this may readily be dissolved by heat ; (iii.) copaiba and other resins give haze in a similar position, but the odour is characteristic ; (iv.) the haze due to the presence of albumoses disappears on heating, and reappears on cooling ; (v.) both pus and blood contain albumen, and if present in the urine, give this reaction, apart from the presence of free albumen.<sup>1</sup>

2. *Boiling.* After testing with litmus, boil the urine, and afterwards add a drop or two of acetic acid. A generalised white precipitate forms on boiling if albumen is present, not dissolved by acetic acid. It is often convenient to boil the upper part of a column of urine, so as to compare it with the lower part.

The *fallacies* of this test are : (i.) *Phosphates* may be precipitated by heat alone if the urine be faintly acid, neutral or alkaline, but the acetic acid dissolves these and increases the albuminous precipitate. (ii.) Insufficient acid added to a highly alkaline urine may result in the presence of an alkaline albumen not precipitated by heat. (iii.) Excess of acid may redissolve the albumen ; undue natural acidity may have the same effect ; all of which prove the usefulness of test-papers. (iv.) In acid urines, a cloud sometimes appears, not on boiling only, as albumen would do, but *after* the acid has been added, due to mucus. (v.) Copaiba and other resins may give a precipitate insoluble in acid, but their odour is characteristic. (vi.) If the urine is not quite clear, it may be necessary to filter it, if boiling the upper part of the tube gives us no information. If turbid from bacteria, add a trace of NaOH, and a deposit of phosphates occurs which carries the bacteria down with it.

3. *Picric Acid test.* Float carefully a saturated solution of picric acid on the urine by a pipette. A precipitate forming at the line of junction of the fluids indicates the presence of albumen. Urates, alkaloids, and albumoses may also be precipitated, but disappear on heating.

The *quantitative estimation* of albumen may be roughly determined by boiling as above and setting aside the test-tube for 24 hours, and reading off the proportion. It may be more precisely calculated by means of Esbach's albuminometer, a tube graduated for measuring the percentage of albumen. Urine taken from 24 hours' collection is poured into the tube up to the mark U, and the reagent<sup>2</sup> is added up to the mark R. The tube is then set aside for 24 hours, and the precipitate falls to the bottom. The level to which this reaches is then noted, and the number on the glass indicates the grammes per litre (or percentage) of albumen present. Another method is the precipitation by boiling (*supra*), washing the precipitate and weighing. *Fallacies.* 1. This method is not reliable if the sp. gr. of the urine is over 1010. The urine should be diluted to 1010 ; and a calculation made afterwards by multiplying the result by the number of times of dilution. 2. If the patient is taking quantities of

<sup>1</sup> The quantity of albumen due to the presence of pus or blood is very slight. Shake up the pus through the urine and count the cells under the microscope. 100,000 pus cells per cub. millimetre cause about 1 per cent. albumen, estimated by Esbach's tube. Blood in sufficient amount to make a urine red gives only  $\frac{1}{2}$  per 1,000 of albumen.

<sup>2</sup> Picric acid, 1 part ; Citric acid, 2 parts ; Water, 100 parts.

alkaline salts, crystals are liable to appear after adding the reagent, and these must be allowed for in reading off the quantity of albumen.

§ 280. **Mucin** is precipitated, as above mentioned, by most of the same reagents as albumen, but it may be detected by taking a saturated solution of citric acid in a test-tube and trickling the urine down the sloped side of the tube, when a cloud forms above the junction of the fluids. Excess of mucus indicates irritation of the bladder or genito-urinary tract; or a vaginal or uterine discharge.

§ 281. **Sugar (glucose)** is not a normal constituent of the urine, but it may occur as a permanent or temporary pathological product. The chief cause of *permanent* glycosuria (sugar in the urine) is Diabetes Mellitus (§ 310). It should be remembered that the sugar may disappear from the urine in this disease for some days, and reappear again as abundantly as before. *Transient* glycosuria is found with errors of diet, excess of carbohydrates, usually in gouty people. Its numerous other causes are referred to in § 309.

TESTS FOR GLUCOSE.—1. *Trommer's test* constitutes one of the readiest for discovering sugar. To an inch of urine in a test-tube add  $\frac{1}{2}$  its volume of caustic potash and a few drops of a solution of copper sulphate. On boiling, a red precipitate denotes the presence of glucose.

2. *Fehling's test*. Fehling's solution consists of an alkaline solution of potassio-tartrate of copper, so prepared that 10 c.c. is reduced by 0.05 gm. of glucose. As it is apt to alter on keeping, it should be boiled before using, to make certain that no precipitate forms before adding the urine.<sup>1</sup> Add to it a few drops of urine and boil again; and then continue adding till equal quantities of urine and Fehling are used. If on further boiling the solution is still clear, no noteworthy quantity of sugar is present. The Fehling must always be in excess, and the boiling must not be too prolonged. This test depends upon the fact that glucose has the property of reducing cupric salts when heated in the presence of a free alkali.  $\text{CuSO}_4$  added to  $\text{NaOH}$  causes a pale blue precipitate of hydrated cupric oxide. If a tartrate is present, the cupric hydrate is held in solution (Fehling's Solution). If glucose or some other readily oxidisable substance is added, this blue cupric hydrate on gently heating is reduced, and falls as a *red* or *yellow* precipitate of cuprous hydrate ( $\text{Cu}_2\text{O}$ ,  $\text{H}_2\text{O}$ ), which on longer boiling becomes red or purple cuprous oxide ( $\text{Cu}_2\text{O}$ ).

*Fallacies*. (i.) The urine to be tested must be freed from albumen, and (ii.) it must not be ammoniacal. (iii.) Other reducing agents may occasionally give the reaction. After the administration of chloroform, chloral, morphia, curare,  $\text{CO}$ , and some other drugs, a reaction is obtained in the urine resembling that due to glycosuria, but it is due probably to the presence of glucuronic acid. Lactose, uric acid and urates, ammonium chloride, and other ammonium salts, hippuric acid, kreatinine, oxyacids and the products of certain drugs, such as carholic or benzoic acids, may occasionally be sources of fallacy. To avoid these it is best to control by the Fermentation Test; or to filter a few drachms of the urine

<sup>1</sup> It is better to keep the copper solution and the alkali solution in separate bottles, mixing them just before using.



through a charcoal filter 7 or 8 times, by which means all reducing substances other than sugar are removed.<sup>1</sup>

*Quantitative estimation by Fehling's Solution.* The urine should be a sample taken from the total collection of 24 hours. Fill a burette with urine diluted to 1 in 20, and have 10 c.c. Fehling's solution in a porcelain dish, diluted with water. Boil the Fehling, and while boiling allow drops of urine to mix with it, stirring all the while. Urine must be run in from the burette till the fluid is colourless; this is difficult to decide unless the dish be tilted so that it shows against the white background apart from the red precipitate which collects at the bottom. Read off the amount of urine required for complete reduction and calculate. Supposing

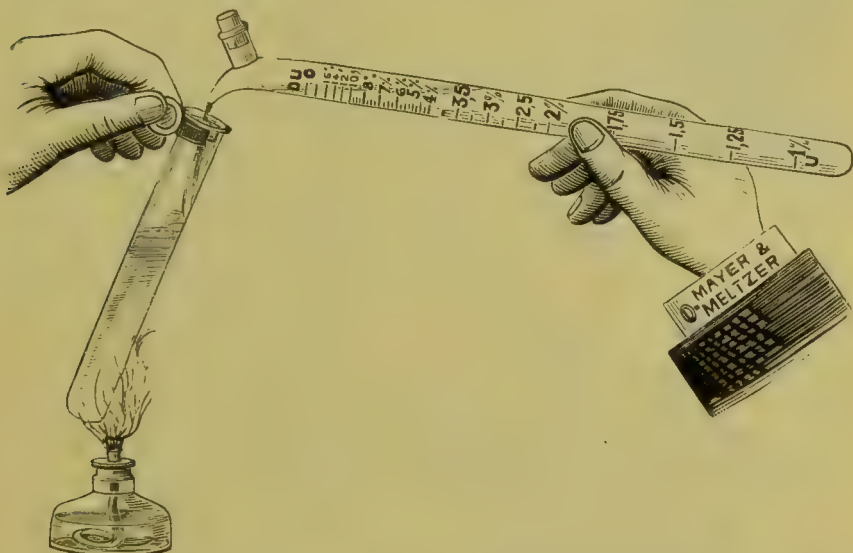


Fig. 77.—CARWARDINE'S SACCHARIMETER. A sample of the 24 hours' collection of urine is used to fill the burette (on right of figure) up to the letter U. Dilute it by adding water to "D U," and mix thoroughly. Fill the measure supplied with the apparatus up to F with Fehling's solution, and dilute it by adding water to "D F." Pour this diluted urine drop by drop from the burette until all the blue colour has gone from the supernatant fluid. This may take some little time as it is necessary after each boiling to wait a minute for the precipitate to subside a little. For calculation see text.

we find that 60 c.c. *diluted* urine from the burette are required to decolourise the 10 c.c. Fehling (representing 0.05 gm. glucose); then  $\frac{60}{20} = 3$  c.c. urine contain 0.05 gm. glucose. Then from this, as we know the number of c.c. urine passed by patient in 24 hours, it is easy to calculate the percentage of sugar excreted in that time. Carwardine's Saccharimeter (Fig. 77) may be employed in this process if an ordinary burette, as used in the laboratory, is not accessible.

2a. *The Ammoniated Cupric Solution* (Pavy's Test) is a modification of Fehling's solution. It contains free ammonia, and this, when cuprous oxide is precipitated by the addition of diabetic urine, keeps the oxide in solution. Hence the *blue colour* of the fluid is discharged *without the formation of any precipitate*, and it is thus easier to determine the exact point when the whole of the cupric salt is reduced than is the case when Fehling's

<sup>1</sup> Saundby, *B. M. J.*, April 14, 1900.

solution is employed. Pavy's solution (10 c.c. of which represent 0.005 gm. of sugar) is therefore very useful as a quantitative test. It is usually applied by means of a special apparatus.

3. The *fermentation test* constitutes the ultimate test in all cases of doubt, since sugar is the only known substance fermented by yeast. After seeing that the urine is acid, fill a test-tube with it, and insert a piece of German yeast; then invert the tube over a saucer of water (or mercury) and place them in a warm place. Have a control tube beside it with normal urine or plain water. If sugar is present, bubbles of  $\text{CO}_2$  gas will form and collect at the top of the tube.

This test can also be applied for the *quantitative estimation* of sugar, by Robert's differential density test. *Method.* Have two 12 oz. bottles with a slit cut in the side of the corks for the gas to escape, and put in each 4 oz. of the urine—taken from a 24 hours' sample. Add a piece of German yeast, the size of a walnut, to one of them and set them aside in a slightly warm place for about 24 hours. Then take the sp. gr. of the two samples; and the difference between them gives the measure of the sugar in grains per oz. The percentage is found by multiplying this difference by 0.23. Thus supposing the sp. gr. of the two samples is 1050 and 1005 respectively; there were 45 grs. of sugar per oz., and  $45 \times 0.23 = 10.35$  per cent. It is important to wait until all fermentation has ceased, and to see that no decomposition of urea has taken place in the control bottle.

4. *Picric acid test.* Boil a few drops of liq. potassæ with a saturated solution of picric acid. Add urine and boil; a dark claret-red colour denotes glucose.

5. *Phenyl-hydrazine test.* To about a drachm of urine in a test-tube add 4 gr. phenyl-hydrazine hydrochlorate and 2 gr. of sodium acetate; boil it in a water-bath for about half an hour. Allow it to cool by placing the tube in cold water. A yellow deposit forms, which under the microscope shows fine yellow crystals, needle-shaped, in clusters.

6. *Nitro-propiol test.* This test is of value for confirming the presence of glucose in urine, when indicated by other reagents such as Fehling's Solution. The reaction is not influenced by uric acid, creatinine, glucuronic acid, bile colouring, or bodies which may be contained in the urine after the administration of certain drugs. When, therefore, urine is found to reduce Fehling's Solution, it is advisable to apply the nitro-propiol test in order to make sure that the reduction is due to glucose. Tablets of nitro-propiol (sodium nitrophenyl-propiolate), each containing gr.  $\frac{1}{2}$  (0.016 gm.), are supplied by Burroughs and Wellcome. Add one to a mixture of 10 minims of urine and 3 drachms of water, and boil. If glucose be present, the liquid will assume a deep blue colour, owing to the formation of indigo blue. If the colour does not appear quickly, continue the boiling for about five minutes before deciding on the result of the test.

**Lactosuria.** Lactose may be present in the urine in considerable quantity, in women who are nursing. Lactose does not answer to the fermentation test, but it does to Fehling's solution. In calculating results, remember that 10 parts of lactose have the same reducing power as 7 parts of glucose.

§ 282. **Urea.** A healthy male adult, weighing, say, 140 lbs. excretes about 3.5 grains of urea per pound of his body weight (0.5 gramme per kilo.). We may say, therefore, in round figures, that he excretes daily about 50 ounces of urine, 500 grains urea (or 10 grains to the ounce), and that the urine contains about 2.3 per cent. of urea<sup>1</sup>—the corresponding figures on the metrical system being approximately 1,320 c.c., 33 grammes, and 2.3 grammes per 100 c.c. But these figures vary widely in health,

<sup>1</sup> It is useful to know that the number of *grains per ounce* multiplied by 0.23 gives the *percentage*.

and are much less (say 300 grains) for a lighter person taking less food. If the kidneys are acting well the urea output may be increased by an increase in the nitrogenous food.<sup>1</sup> On the other hand, it is considerably diminished after vomiting or diarrhœa. Particulars on all of these points should be investigated and noted; and a *specimen for estimation should be taken from the urine of 24 hours, mixed and measured* (§ 278). Finally, several such observations should be made before concluding that there is really deficient nitrogenous elimination, or that the renal adequacy is impaired. There is a deficient elimination of urea sooner or later in nearly all renal diseases (the accompanying effect being uræmia, § 273), in certain hepatic diseases, in myxœdema, Addison's disease, and melancholia.

**ESTIMATION OF UREA.** The specific gravity of the urine gives us (in the absence of sugar) a very fair idea of the quantity of urea being excreted; indeed, that is the chief reason why we habitually use the urinometer (§ 276). The rapid crystallisation of nitrate

of urea in a test tube when an equal bulk of strong nitric acid is added to the urine and the mixture cooled, suggests excess; but for accurate results it is necessary to determine the *total nitrogen* in the urine (the greater portion of this being in the form of urea) by volumetric analysis. This has been now rendered available for clinical practice by the simple apparatus described below. Albumen, if present, should be separated by boiling and filtration before beginning the estimation of urea; and the specimen of urine should be from the 24 hours' collection mixed.

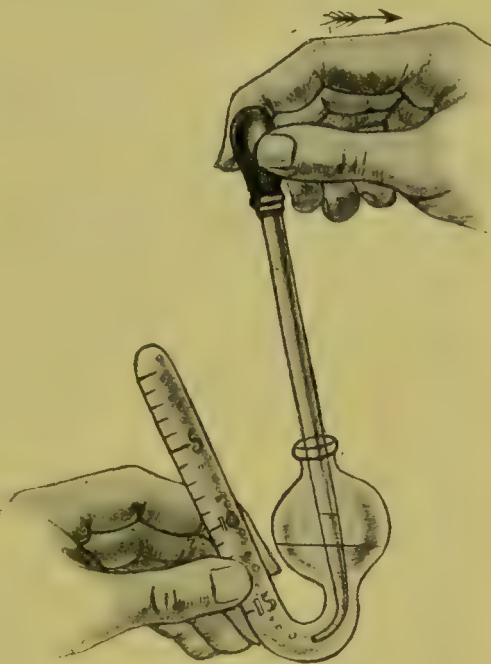


Fig. 78.—DOREMUS'S UREAMETER.

<sup>1</sup> The diurnal quantity of urea depends partly upon the destruction of nitrogenous tissues in the body, but chiefly upon the amount of proteid ingested. The urea dependent upon the latter for its source is usually accompanied by a parallel increase in the sodium chloride in the urine, because most proteid foods are rich in sodium chloride. If, however, the urea which depends on tissue-destruction for its source, be increased, it is unaccompanied by a parallel increase in the sodium chloride. It is sometimes in practice a little difficult to determine whether deficiency of urea depends upon deficient tissue-destruction or deficient intake of proteids. But if the physiological facts just named can be relied upon, we have in the estimation of the chlorides an answer to the question.

Doremus's Ureometer (Fig. 78) is so easily used that it can be employed for estimation in one's consulting room. There are two stages in the process. (1) To fill the vertical U tube with a solution of hypobromite of sodium. This must be freshly prepared immediately before use, and it is best to keep it in two solutions, potash in one, bromine in the other, as suggested by Martindale ("Prescriber's Companion"), to be mixed in equal parts just before using. The bromine odour is so powerful that the bottle containing it can only be kept in a bathroom or lavatory. First, holding the tube vertically, the operator pours the solution into the bulb, until it is about half full, then he inclines the apparatus horizontally so that the fluid passes up into the long or vertical limb of the U tube; then he restores it to the vertical position and repeats the process until the *vertical limb is quite full*, and the bulb is one third full or thereabouts. The tube now contains about 35 c.c. (2) The second stage requires considerable exactitude. The accuracy of results depends upon the care with which the pipette is manipulated. Having drawn up the urine *very precisely* to the 1 c.c. mark on the pipette, wipe the outside of the pipette rapidly with a towel, and introduce it as shown in the figure just beyond the bend of the U tube. Now comes the most ticklish part of the operation. The rubber top is gently squeezed so as to *SLOWLY*, and *CONTINUOUSLY*, press out the urine. *Watch the tip of the pipette carefully* as the urine slowly passes out so as to prevent (i.) the hypobromite from coming back into the pipette; and (ii.) the air from the pipette going out into the hypobromite. If either happens, the whole must be done again. The nitrogen, which is rapidly liberated and collects in the upper end of the vertical limb of the U tube, is the exact measure of the urea contained in the 1 c.c. of urine used. Set the tube carefully for an hour until the bubbles and heat evolved have subsided, then read off the percentage of urea, or grains per ounce, as marked on the side of the vertical limb. The urine must be examined fresh, *and if it contains albumen this must be removed by boiling and filtration*. The U tube may be held in the hand as shown, but it should be rested on a table or stand. This process estimates the uric acid and the other products of nitrogenous disintegration as well as the urea, but this does not invalidate the process for clinical purposes.

§ 283. Uric (*i.e.*, lithic) acid, either free or combined in the form of **urates**, is normally present in a sample from a day's collection to the extent of .04 per cent., or about 8 or 9 grains per diem. According to Dr. Alexander Haig, it bears in health a fairly constant proportion to the amount of urea, 1 grain of uric acid per day (per 10 lbs. body-weight) to 35 grains of urea per day (per 10 lbs. body-weight); the two rising and falling together. Others (*e.g.*, Dr. A. P. Luff) say the proportion is about 1 to 50. Uric acid and urates when in excess are best detected as a cloudiness or deposit (§§ 290 and 291). Their chemical quantitative estimation is a matter of some delicacy and difficulty.

The murexide chemical test for uric acid, free or in combination as urates, is performed by adding nitric acid to the suspected deposit in a porcelain dish, heating to dryness, and placing a drop of ammonia on another part of the dish. Where the two join, a characteristic purple coloration appears. If a drop of caustic potash be placed on another part of the dish, a blue coloration appears at the junction. To accurately estimate the quantity of free or combined uric acid in the urine, Gowland Hopkins' method is usually employed, or that of Hayeraft, both of which are laboratory methods.

Gowland Hopkins' Method for the quantitative estimation of uric acid free and combined in urine is thus summarised by Dr. A. P. Luff ("Goulstonian Lectures on the Chemistry and Pathology of Gout," *The Brit. Med. Journ.*, 1897, vol. i., p. 771). "Saturate the urine with powdered ammonium chloride, allowing it to stand for 2 hours, with occasional stirring and then filtering. The precipitated ammonium urate is washed three or four times with saturated ammonium chloride solution, washed off the filter into a small beaker with a jet of hot distilled water and heated just to boiling with excess of hydrochloric acid. The beaker and contents are allowed to stand in the cold for 2 hours, when the uric acid separates out completely, and is collected on a filter and washed with cold distilled water. (The filtrate should be measured before washing, and 1 m. added to the final result for each 15 c.c. of filtrate.) The uric acid is washed off the filter with hot water, warmed with sodium carbonate till dissolved, then transferred to a flask and mixed with 20 c.c. of pure sulphuric acid. The mixture is titrated warm with a twentieth normal solution of potassium permanganate till a permanent pink colour is obtained; 1 c.c. of the permanganate = 0.00375 gr. uric acid." The chief precaution to observe in this process is to wash your precipitates very thoroughly. If that be done, the method is extremely accurate.



§ 284. **Bile** is present in the urine in cases of jaundice, and it can be detected there even before the skin assumes a yellow colour. Both bile pigments (especially bilirubin) and bile acids are present, the former more abundantly. An orange-green colour of the urine betrays the presence of bile if in any quantity.

(i.) *Gmelin's test* for the bile pigments: Add a drop of nitric acid to the urine on a porcelain slab, and a play of colours will be seen where the fluids meet. (ii.) *Marchal's test*: Add a few drops of tincture of iodine to the surface of the urine in a test-tube by means of a pipette, and a green reaction is obtained. (iii.) *Pettenkötter's Test for Bile-acids*: Add a solution of cane-sugar to urine: pour strong  $\text{H}_2\text{SO}_4$  down the side of the glass. At the junction line a cherry-red colour appears. This is useless unless the urine contains a considerable quantity of bile. The urine must also be free from albumen.

*Bilirubin* and *Hæmatoidin* have very occasionally been found free, as a deposit, in the urine in a granular or crystalline form in jaundice, acute yellow atrophy, carcinoma of the liver, scarlatina, typhoid, or after a hæmorrhage or the bursting of an abscess into the urinary passages. They both occur as amorphous, orange-red granules, or as crystals of the same colour, shaped like needles or rhombic plates. Both give a green ring with nitric acid. Hæmatoidin may be distinguished by forming a transitory blue colour with nitric acid, and being insoluble in caustic potash and ether, in which reagents bilirubin is soluble.

§ 285. **Blood** in the urine (*Hæmaturia*) when in any quantity imparts to the urine a characteristic "smoky" colour, and red blood cells may be identified under the microscope (§ 292). A dark colour of different shades may also be imparted to the urine by *Methæmoglobinuria*, *Hæmatoporphyrinuria*, *Alcaptonuria* (all of which are referred to below), and *Carbolic Acid*. The most delicate test for hæmoglobin, either free or combined in the corpuscles, is the spectroscope (see Chapter XX.).

*Chemical test for blood*.—Add a few drops of freshly-prepared tr. guaiaci to the urine and shake, then add excess of ozonic ether. A blue line appears at the junction of the fluids. The same reaction may be obtained by using filter- or blotting-paper. Allow a drop of each of the reagents to fall on the paper beside a drop of the urine, noticing the colour at the junction of the three drops. *Fallacies*. Saliva gives the same reaction, and so do iodides, in patients taking these salts. Pus gives a blue colour with guaiacum alone. It is very important to have the tinct. guaiaci freshly prepared, and to this end it is best to dissolve a morsel of the resin in S. V. R. at the time when it is used.

**Hæmoglobinuria** is always present with hæmaturia, because the corpuscles break up. Its presence *alone* is rare, and can only be proved by examining the centrifugalised deposit of absolutely fresh urine under the microscope and finding *no red cells*.

**Methæmoglobinuria**. The characteristic smoky colour of the urine in hæmaturia of renal origin, depends largely on methæmoglobin, a substance formed from hæmoglobin by the action of acid urine. It is this pigment also which is found in *Paroxysmal Hæmoglobinuria*. It is recognised by the spectroscope.

**Hæmatoporphyrinuria** (Iron free hæmatin in the urine) is a condition where the urine has a dark cherry-red colour like port-wine, but gives no guaiacum reaction. It is found after excessive dragging with sulphofal, and is an indication for at once stopping the drug and giving alkalis freely. It is known by its spectroscopic bands. If these

cannot be detected in the urine, the hæmatoporphyrin should be extracted by shaking with acetic ether or amylic alcohol, after adding a few drops of acetic acid; the extract so obtained will give the four characteristic bands.<sup>1</sup>

§ 286. Pus in the urine is best detected by the microscope (§ 292).

When in considerable quantity it may be detected chemically by the addition of an equal quantity of liq. potassæ to the deposit. A ropy gelatinous mass is formed, which pours from one test-tube to another like a fluid jelly. This test is only applicable when a fair quantity of pus is present. In small quantities it is best to make a microscopic examination of the deposit for pus cells. When pus comes from the *kidney* the urine is, at any rate when first passed, acid, and the pus is *uniformly disseminated* through the urine, and remains so for some time. When it comes from the *bladder*, the urine is alkaline, or neutral, and the pus very rapidly collects into a *creamy layer* at the bottom of the glass.

§ 287. Salts in the urine. CHLORIDES. The chlorides found in the urine are principally salts of sodium; and vary in *health*, according to the food taken, from about 11 to 15 grms. daily. In *disease*, the chlorides are increased during convalescence from fevers, during the stage of absorption of œdema or other forms of serous exudations and in diabetes insipidus. They are diminished in acute fevers, especially pneumonia (reappearing at the crisis), in renal diseases with albuminuria, in gastric disease, such as cancer or dilatation, where the digestive power is diminished, in anæmic conditions, and, it is said, in melancholia, idiocy, and dementia.

*Test.* Add a few drops of  $\text{HNO}_3$  to the urine, and an equal bulk of 3 per cent. sol.  $\text{AgNO}_3$ . A curdy precipitate follows if the chlorides are normal in quantity; if the urine only becomes milky, they are diminished.

*Quantitative estimation of Chlorides* (Mohr's Method). After the urine has been freed from albumen, take 10 c.c. and mix with it 50 c.c. of distilled water; then add a pinch of calcic carbonate and 3 drops of a neutral chromate of potassium solution (1 in 20). The calcic carbonate neutralises any free acid which may be present. To this a standard solution of silver nitrate is slowly added from a burette, stirring constantly. The white precipitate of chloride of silver separates first, but the silver nitrate solution must be added drop by drop until the faintest tinge of pink appears. The pink colour is an indication that chromate of silver is now being formed, all the chlorides having first united with the silver.

*Calculation.* Take the total number of c.c. of silver nitrate used, and deduct 1 c.c. to account for other substances present in urine which unite with the silver; then every remaining c.c. of the solution used represents 10 milligrammes of sodium chloride.

PHOSPHATES. Phosphates in excess may be attended with a group of somewhat vague symptoms, which are elsewhere described (§ 315).

*Tests.* In an *alkaline* or neutral urine phosphates spontaneously form a cloudy precipitate, which is increased on boiling, but which disappears on acidifying the urine. If present in a urine that is already alkaline, the deposit is distinguished from pus by the fact that it is dissolved by acetic acid. The microscope enables us to distinguish between pus and phosphates with certainty (§ 292); this instrument is indispensable when, as often happens, the two deposits occur together. If the urine be *acid*, it is necessary first to add some caustic potash; and if it be then heated the phosphates are precipitated.

SULPHATES are also normally present in the urine, but their excess or insufficiency has no clinical significance.

§ 288. Proteids in the urine. Besides serum albumen, hæmoglobin, methæmoglobin, and hæmatoporphyrin, the only proteid which in the present state of our knowledge has any clinical significance is albumose. Mucin and nucleo-albumen are derived from the urinary passages, and in

<sup>1</sup> Readers who are interested in the subject of proteids and pigments in the urine should consult the writings of Dr. Archibald Garrod (*Journ. of Physiology*, 1894, xvii., p. 349; and in the Bradshaw Lectures, *The Lancet*, Nov. 10, 1900).

such small quantities as to be negligible. For further details the reader is referred to larger works on the subject.<sup>1</sup>

**Albumosuria** was formerly known as **Peptonuria**, but it is now supposed that true peptones never appear in the urine. Albumosuria occurs where there is great destruction of white corpuscles, and therefore whenever there is a large collection of pus in the body, *e.g.*, in empyema and any abscess formation. It is useful in deciding the character of an effusion, pleural, peritoneal, or meningeal. Albumosuria also occurs whenever tissue destruction takes place under the action of micro-organisms. It appears in the resolution stage of pneumonia, and has been described in connection with osteomalacea, with certain liver diseases, such as acute yellow atrophy, with ulceration of the intestine, with dyspepsia, sometimes when excess of animal food is consumed, and finally with some cases of nephritis (together of course with albuminuria).

*Test.* Primary and secondary albumoses are found—the latter being more nearly related to the peptones—but they have the same clinical significance. (1) Presuming the urine to be free from ordinary albumen, add  $\text{HNO}_3$  drop by drop to the urine; if a precipitate is formed which disappears on heating and reappears on cooling, *primary* albumoses (? peptones) are present. Both forms of albumose react to the next test. (2) Acidify the urine strongly with acetic acid, add an equal bulk of saturated salt solution, till a cloud forms; if it disappears on heating and reappears on cooling it is due to albumose.

§ 289. Other rare constituents in the urine are acetone, diacetic acid, dioxyphenyl acetic acid, and indican.

**Acetonuria.** An infinitesimal trace of acetone is always present in the urine, and this may be increased by a highly albuminous diet. Acetone is also increased in some febrile states, in cancerous cachexia, and other conditions of inanition, because of the increased albuminous destruction in such states. It was formerly believed that the presence of a definite quantity of acetone in the urine heralded in diabetic patients an attack of diabetic coma, but the evidence of this is not very satisfactory.

*Test.* Make a solution of gr. xx. pot. iod. in 5i liq. potassæ. Warm gently, add an equal quantity of urine; a precipitate of iodoform crystals (hexagonal plates) forms if acetone is present.<sup>2</sup> Acetone reduces Fehling's solution.

**Diacetic acid** is found in the same conditions as acetonuria. Its presence is detected by adding a few drops of a strong solution of ferric chloride, when a deep red colour appears.

**Alcaptonuria** is a condition where the urine forms a pellicle on the surface and darkens from the surface downwards on standing exposed to the air, due to the presence of dioxyphenyl acetic acid. It has no known clinical significance. Its only importance lies in the fact that it reduces Fehling's solution.

**Indicanuria.** Indican (indoxyl sulphate of potassium) is found—(1) where there is an excess of intestinal putrefaction, hence it is present also in cases of gastric disorder with deficient  $\text{HCl}$ ,  $\text{HCl}$  being an antiseptic agent. (2) In peritonitis, and some other diseases where the peristalsis of the small intestines is impeded. Some maintain that in simple chronic constipation without intestinal obstruction there is no indicanuria, but von Jaksch<sup>3</sup> and others affirm its presence in constipation and all gastric disorders. (3) With empyema, putrid bronchitis, etc. (4) Indican is also said to be present, in small quantity, in decomposing urine. *Test.* Add an equal bulk of strong fuming  $\text{HCl}$ , and a few drops of a solution of hypochlorite of lime (bleaching powder), to the urine. This on standing for a few minutes produces a blue colour due to indican, which may be extracted by shaking up with about one-third its volume of chloroform.

### c. The Urinary Deposit.

§ 290. Cloudiness of the urine (naked eye examination). In healthy urine there is no deposit, but most of the normal constituents if in excess, and some abnormal substances, become evident as a sediment or turbidity. (1) A bulky pinkish turbidity and

<sup>1</sup> See an interesting Discussion on this subject at the Roy. Med. and Chir. Soc. of London, *The Lancet*, 1900, vol. 1.

<sup>2</sup> It is generally believed among chemists that this reaction, ascribed to acetone, is really due to diacetic acid.

<sup>3</sup> *Loc. cit.*

deposit in an acid urine, which forms when the urine cools, indicates the presence of *lithates*—i.e., urates. It is the commonest of urinary deposits, and its appearance when the urine gets cold is quite typical of lithates. (2) *Uric acid* is evident to the naked eye as a sandy deposit resembling red cayenne pepper. (3) A white flocculent turbidity in an alkaline or neutral urine indicates the presence of *phosphates*, which are cleared at once by the addition of a few drops of acetic acid. (4) *Calcium oxalate* gives a typical “powdered-wig” deposit of fine white points seen on the surface of a mucus cloud. (5) A fine cloud of *vesical mucus* is normally present in the urine, although it is only visible when the entangled mucus and epithelial cells are sufficiently plentiful. (6) *Pus* forms a deposit which resembles phosphates to the naked eye, but it is readily distinguished under the microscope. (7) Urine is sometimes cloudy from the presence of *bacteria*, and this cloudiness cannot be cleared by boiling or the addition of acids.

§ 291. **Microscopic specimens** of the deposit must always be examined in cases of suspected renal disease. The urinary deposit is best examined after the urine has stood for some hours in a conical glass; or after the specimen has been centrifugalised.<sup>1</sup> Take a pipette, close it at the top with the right forefinger, pass it to the bottom of the glass, allow a small quantity of the sediment to enter, withdraw the pipette, wipe its exterior with a cloth, place the point on a microscopic slide, then surround the pipette with the palm of the left hand, the warmth of which will cause a drop to exude. Cover the drop with a cover-glass and examine first, under a  $\frac{1}{3}$ rd or  $\frac{1}{2}$  inch objective, then under a  $\frac{1}{4}$ th or higher. The deposit normally contains foreign substances, such as cotton and woollen fibres, etc., and a few bladder (and in women nearly always a few vaginal) epithelial cells, which are recognised by their large and nucleated appearance. Inquiry should always be made as to the sex of the patient, and in women if any leucorrhœa is present. If so, it is very desirable to draw off a specimen of urine by the catheter.

The urinary deposit may contain ORGANISED SUBSTANCES (§ 292), or CRYSTALLINE or unorganised substances (§ 293).

§ 292. The **organised constituents** of the urinary sediment are of far more serious import than the crystalline substances. They comprise TUBE-CASTS (which are the most important), EPITHELIAL CELLS, PUS CELLS, BLOOD CELLS, spermatozoa, and certain rarer substances such as microbes, fat cells, etc.

**Tube-casts** and renal **epithelial cells** are present in all renal

<sup>1</sup> For centrifugalisation a special apparatus is necessary, the specimen being placed in a tube on the edge of a rapidly rotating wheel. It is accurate, convenient, and saves time.



maladies attended by disease or destruction of the renal epithelium. When tube-casts are abundant in the urine microscopic examination of the sediment readily detects them. But if, on the other hand, they are present only in small numbers, they may be readily overlooked, and this all the more easily occurs when, as in chronic interstitial nephritis and in amyloid disease, the urine is abundant and of low specific gravity, so that any suspended matter it contains is collected as a deposit only slowly and incompletely. Further, these are the exact instances in which the casts are apt to be of the hyaline variety, and their more or less transparent character renders them inconspicuous objects in the microscopic field. Hence the search for tube-casts must be conducted with great care if the risk of a false conclusion is to be avoided. One of the best methods, after settlement or centrifugalisation of the deposit, is to examine it with a moderately low power of the microscope, using a narrow diaphragm and shading the light so as to have the field only feebly illuminated. Any suspicious-looking object can be brought into the centre of the field and examined with a stronger lens. In this way casts may be detected which in a strong light would readily be missed, and if several slides have been prepared and examined in this manner the detection of any casts present in the urine is rendered fairly certain. But the examination should be repeated on several occasions in any urine containing albumen before a negative conclusion is finally arrived at. There is often a special degree of difficulty in finding casts in alkaline urine, and in decomposing urine they undergo disintegration.

The clinical importance of tube-casts arises from the fact that with but few exceptions their presence in the urine definitely indicates disease of the renal epithelium. Thus, when found in a urine containing albumen they add great weight to the opinion that the albuminuria is a result of some structural change of the kidney. Similarly in cases of pyuria and hæmaturia respectively the detection of tube-casts not only suggests that the pus and blood are of renal origin, but also that the kidney is becoming involved. It must be remembered that more than one part of the urinary tract may be diseased at one and the same time. In the urine of patients who are jaundiced, tube-casts may often be found without, either at the time or subsequently, any evidence of renal disease.

Regarding the different kinds of casts (Fig. 79), these are to a large extent intermingled in most cases. But, in general terms, *epithelial casts* and *blood casts* are indicative of the earlier and more acute stages of parenchymatous nephritis. *Hyaline* or waxy casts are not peculiar to lardaceous kidney, but occur in other forms of both recent and long standing renal disease. These and *fatty casts* indicate that the inflammatory process is passing to a degenerative stage. *Granular casts* are



Fig. 79.—RENAL TUBE-CASTS—*a*, epithelial casts; *b*, granular cast; *c*, hyaline casts; *d*, fatty cast; *e*, blood casts.

more abundant in chronic renal disease, both tubal and interstitial. Tube-casts in abundance always form a serious symptom, but a single cast may occur in normal urine. They are more abundant in the acute than the chronic forms of renal disease. Their *absence* does not count for very much as they may be easily missed, or undergo disintegration in the urine.

**Renal Epithelium** (Fig. 80). The detection of renal epithelium in a urinary deposit has much the same significance as the presence of tube-casts. The cells are *spherical* and rather smaller than

bladder or vaginal epithelium. They may be seen isolated or in small groups. In acute Bright's disease they may be found in a fairly normal condition, but in diseases of a chronic order they become much changed, and may thus appear crowded with fat globules. **BLADDER OR VAGINAL EPITHELIUM** (Fig. 81) is met with as collections of squamous cells; transitional, spindle-shaped, and other forms of epithelium may also be derived from the bladder. **TAILED EPITHELIUM** may be derived from the pelvis of the kidney, and the presence of cells having this elongated character would greatly aid the diagnosis in a case of suspected pyelitis. It must be remembered, however, that the male urethra and the prostate gland yield epithelium practically identical with the above. A deposit from this source is not uncommon in cases of chronic prostatitis the result of a former gonorrhœa.

**Pus Corpuscles** (leucocytes in the wrong place) under the microscope, are

of globular form with a diameter about one-third larger than that of a red blood-cell. The corpuscles are opaque and granular, but when treated with acetic acid they clear up, and a nucleus, often partially split into two or more divisions, is seen (Fig. 82, *a* and *b*).

**Red Blood Corpuscles.** The detection of red blood corpuscles in a urinary deposit is, of course, conclusive evidence of the presence of blood. In most fresh urines they are readily distinguished, as they retain their bi-concave form and the outline shows a double

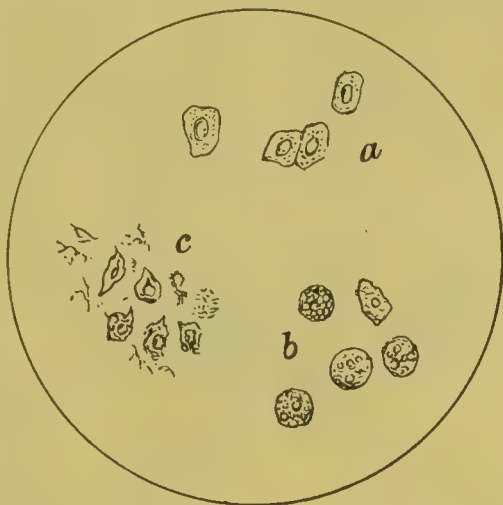


Fig. 80.—RENAL EPITHELIUM—*a*, normal; *b*, fatty; *c*, disintegrating.

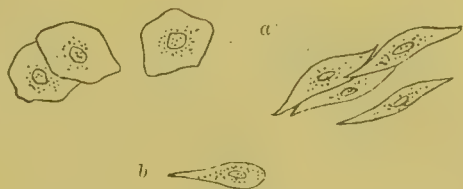


Fig. 81.—BLADDER EPITHELIAL CELLS (*a*); and, TAILED EPITHELIUM (*b*) from the pelvis of the kidney.

contour (Fig. 82, *c*). But sometimes the corpuscles become much changed. Thus in a very dilute urine they are apt to become distended by imbibition, and then are seen as circles having sharp delicate outlines (*d*). In other instances they become crenate, shrunken, and deformed (*e* and *f*).

**Spermatozoa** may occasionally be found in the urine. Each has a minute oval or pear-shaped head, from the larger extremity of which

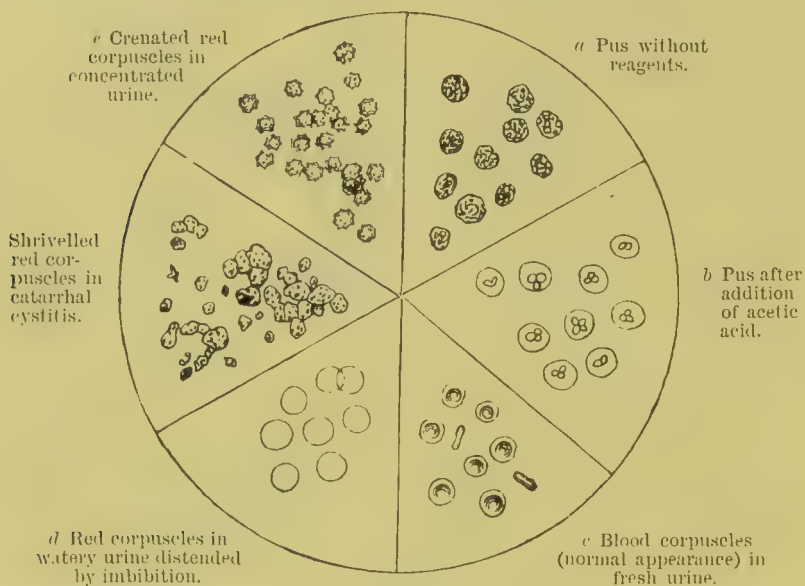


Fig. 82.—Various appearances of RED BLOOD CORPUSCLES and PUS CELLS. In very pale watery urine, the red corpuscles may be so pale as to escape detection (*d*). They may then be revealed by adding a solution of iodine in potassium iodide.

there passes a long and delicate tail. The total measurement of the spermatozoon is about  $\frac{1}{800}$  inch in length.

**Microbes.** Numerous microbes are found in the urine, especially when decomposition has occurred either within the bladder or subsequently. The most constant are the *bacillus ureæ*, *vibriones*, and the *hay bacillus* (*bacillus subtilis*), which have no special clinical significance apart from putrefactive changes. *Gonococcus* is found in cases of gonorrhœa; and the *typhoid bacillus* is very abundant in cases of enteric fever.

The TUBERCLE BACILLUS may be found in tuberculous disease of the bladder or pelvis of the kidney, and is a sign of great value. In appearance under the microscope it resembles the smegma-bacillus and other organisms. Its special staining reaction will be given in Chapter XX. It is difficult to find in the urine early in the disease, and in obscure cases the experimental test upon guinea-pigs should be employed, the urine for inoculation being collected through a sterilised catheter into a sterilised bottle.



The *B. COLI COMMUNIS* is sometimes found in the urine in pure culture, and there seem good grounds for the belief that it may produce an Ascending Pyelo-nephritis (§ 306). The bacilli of the urine and their clinical and pathological relations would well repay study.

§ 293. Crystalline and inorganic deposits in a urinary deposit are usually of less serious import than the organised substances above noted.

IN ACID URINES we meet chiefly with urates, uric acid, oxalates, and — among the rarer substances — stellar phosphates, cystin, xanthin, hippuric acid, tyrosin, and leucin.

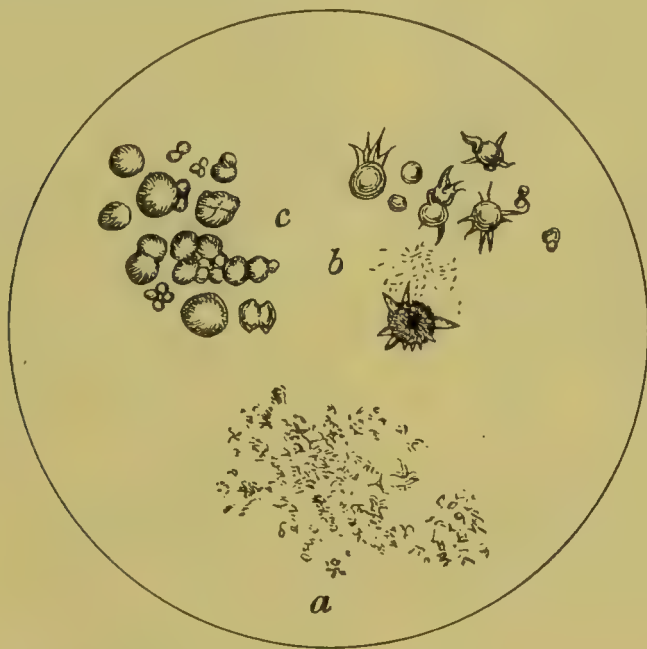


Fig. 83.—URATES. *a*, Amorphous urates of sodium and potassium. *b*, "Hedgehog" crystals of sodium urate. *c*, Ammonium urate.

IN neutral or ALKALINE URINES we meet chiefly with triple phosphates (occasionally urate of ammonium and calcium carbonate).

Amorphous deposits of urate of potash or ammonia, and phosphates and carbonates of the alkaline earths may be met with in urines of EITHER REACTION.

1. URATES (*i.e.* LITHATES), chiefly of sodium, potassium, or ammonium, when in excess are deposited as an amorphous deposit after the urine (warm when first passed) has become cold. A deposit having these characters, and disappearing when heated in a test-tube, is sufficiently

characteristic for the detection of lithates. The deposit is dissolved on the addition of caustic potash; a test which also distinguishes urates from phosphates. *Urates of Soda and Potash*, under the microscope, appear as amorphous orange or *pale brown* granules (Fig. 83, *a*). *Urate of soda* may occasionally appear as "hedgehog" crystals, globular masses covered with spikes (Fig. 83, *b*). *Urate of Ammonium* occurs as globular masses, sometimes spiked, very like sodium urate, but known from such by being found in alkaline urines (together with phosphates) and by being dissolved by acids (Fig. 83, *c*).

Clinically, urates and uric acid are important only when they occur *constantly*, in fresh urine, or in urine that has stood a few hours only. Gout and other symptoms are apt to arise in such cases (see LITHÆMIA.

§ 251), and calculus might be expected to form in the bladder or kidney. An occasional deposit of urates, or a deposit occurring in urine that has stood over 6 hours, is of but little importance. In all concentrated urines, on cooling, large deposits of urates normally occur.

2. FREE URIC ACID is deposited when the urine is very acid, poor in salts and in pigment, and is therefore found chiefly in dilute urines, pale, with deficiency of salts. The red deposit of uric acid closely resembles cayenne pepper to the naked eye, and by patients is sometimes mistaken for blood, but the difference is at once apparent under the microscope. It may be detected in the urinary deposit under the microscope by the *colour* and shape of the crystals. It occurs in the form of *red-brown crystals* (the only coloured crystals commonly found in the urine), mostly lozenge-shaped (Fig. 84). Uric acid assumes many different shapes, owing to the presence of the colloid substances in

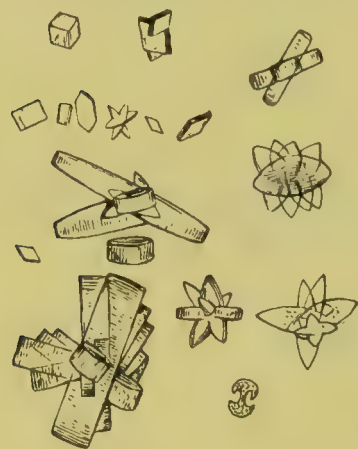


Fig. 84. — URIC ACID crystals (red brown). The two top rows show, from left to right, the evolution in a colloid medium of the "lozenge-shaped" crystal from the primary rhombic prism. In the lower right hand corner is the "dumb-bell" form occasionally met with.

the urine, but they are all derivatives from the rhombic prism or parallelogram, in which form uric acid crystallises from pure water. The more pigment, mucus, and other colloids there are in the urine the more spherical do the crystals become. Some of these are shown in the accompanying illustration, and the gradual transition from rhombic prism to dumb-bell and other spherical forms will be seen by following the crystals from left to right. This deposit is soluble in caustic potash, insoluble in dilute acetic acid, the converse of phosphates.

In health uric acid is increased with a highly nitrogenous diet, after much exercise, after meals, and during the "alkaline tide" of the morning. It is also increased in most fevers, in splenic diseases, pernicious anæmia, in some cases of dyspepsia, during and after an acute attack of gout, and during an attack of acute rheumatism. It is diminished in chronic gout, especially just before the acute exacerbations; in chronic Bright's disease; in chlorosis and other chronic diseases.

3. PHOSPHATES occur as a white deposit or flocculent turbidity in feebly acid, NEUTRAL, or ALKALINE urine, in three different forms, which in order of frequency are :—(1) *Amorphous phosphates of calcium* form the thick white deposit that is apt to be mistaken for pus, but which is more readily shaken up in the urine. These and all other phosphates are soluble in acetic acid, and precipitated by ammonia. The latter test decomposes neutral phosphates. (2) *Triple phosphate* of ammonium and magnesium (Fig. 85), is found in urine which has undergone alkaline fermentation. The crystals are large colourless three-sided prisms like "house-tops," occurring singly, or as snow-flakes or other irregular forms. In markedly ammoniacal urine "feathery phosphates" are found. (3) *Basic magnesium phosphate* occurs in large rhombic plates, not grouped, but scattered (Fig. 86). (4) *Neutral or dicalcium phosphate* occurs in

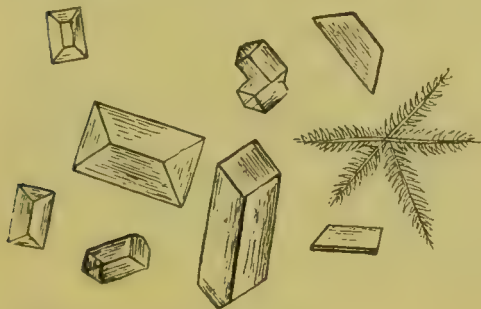


Fig. 85.—TRIPLE PHOSPHATE—"house-top" and "feathery" crystals.

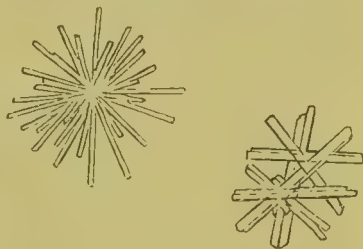


Fig. 87.—NEUTRAL or "STELLAR" PHOSPHATE.

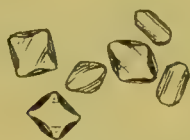


Fig. 86.—BASIC MAGNESIUM PHOSPHATE.

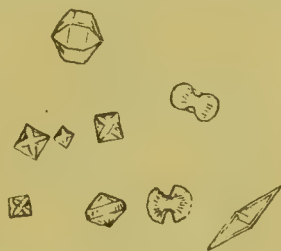


Fig. 88.—CALCIUM OXALATE—"envelope" and "dumb-bell" crystals.

neutral or alkaline urines as clear, refractile, pointed or wedge-shaped prisms arranged in stellate groups—"stellar phosphates" (Fig. 87). The constant presence of phosphatic deposits may be associated with symptoms (§ 315), or suggest the presence of a stone. *Monocalcium phosphate* occurs chiefly in acid urines.

4. OXALATES are chiefly met with as *oxalate of calcium*. This occurs as a scanty crystalline deposit of colourless transparent octohedra, appearing, under the microscope, like tiny envelopes, hence the name "envelope crystals" (Fig. 88). They sometimes rest like fine powder above a cloud of mucus, and have been described therefore as the "powdered wig" deposit. They are soluble in hydrochloric acid, insoluble in acetic acid or caustic potash. Oxalate of calcium may also occasionally appear as dumb-bell shaped crystals. The presence of crystals of oxalate of calcium in the urine is indicative of an excess (OXALURIA, § 315); their presence may also suggest the nature of a calculus.

5. *Calcium Carbonate* is a rare deposit, consisting of tiny spheres and dumbbells, or of amorphous granules, effervescing and dissolving in acetic acid (Fig. 89). The *Carbonates of the Alkaline Earths* are very occasionally found as tiny amorphous granules or concretions. Calcium sulphate and carbonate may take part in the formation of vesical calculi, especially in the aged, but otherwise they are of no clinical significance. Their presence only points to the existence of a calculus, and indicates its composition.



Fig. 89.—CALCIUM CARBONATE.

When a patient is taking crystalline drugs, such as acetate of potash, phosphate of soda, or even liquor ammoniæ, various crystals which have no pathological significance sometimes appear in the urine. Moreover, after a reagent has been added to urine (*e.g.*, Esbach's solution for the estimation of albumen), and it has been set aside, crystals may also appear which have no clinical value.

6. *Certain rare and less important deposits*, which occur chiefly in acid urines, are as follows:—*Hippuric Acid* is an antecedent of uric acid in the nitrogenous metamorphoses of the tissues. It occurs as four-sided prisms, either scattered or in groups. It is present after the ingestion of benzoic acid in large doses, cranberries and other fruits. *Calcium Sulphate* occurs either as amorphous granules, or, very rarely, as long colourless needles or elongated tables with truncated ends. It is detected by being insoluble in ammonia and acids. *Leucin* occurs as laminated spheroids, and *Tyrosin* as bundles of acicular crystals (Fig. 78A). Both occur in the urine in phosphorus poisoning and acute yellow atrophy of the liver. *Cholesterolin* (Fig. 73) is only occasionally found among urinary deposits. It forms laminated plates with longitudinal striae, and a notch at one end. *Cystin* occurs as hexagonal plates soluble in ammonia (Fig. 73A, p. 437).

#### PHYSICAL EXAMINATION OF THE KIDNEYS.

§ 294. A dull "sickening pain" is usually felt on firmly compressing the kidney with both hands, but there is no tenderness in a healthy organ. Tenderness may be elicited in cases of calculous and other causes of pyelitis, perinephric inflammation, abscess or tumour of the organ. Kidney tumours tend to grow forwards, where there is least resistance, pushing the resonant colon *in front* of them. When therefore, the palpating hand encounters resistance and swelling in the lumbar region *posteriorly*, it is probably due to a peri- or extra-renal, rather than to a renal condition (see Fig. 39, p. 165). The diagnosis of renal swellings from other abdominal tumours has been given in § 189.

In the majority of renal disorders the physical examination of the kidneys is of secondary importance to the examination of the urine. The kidneys are situated on either side of the spine, about three inches from the middle line; the right is slightly lower than the left, owing to the position of the liver just above it. The upper end of the r. kidney reaches to the *lower* edge of the 11th rib; the l. kidney reaches as high as the *upper* edge of the 11th rib. The kidneys lie partly in the hypochondriac and partly in the lumbar regions, and are therefore much higher than is commonly supposed, with reference to the anterior abdominal wall. The



lower end of the r. kidney is 1 inch and that of the l. kidney is  $1\frac{1}{2}$  above the level of the umbilicus.

**Palpation.** Even in normal conditions the lower border of the r. kidney may be palpable in thin people. In those whose abdominal wall is lax, in women who have borne children, for instance, it is surprising how frequently the r. kidney can be palpated. The patient should lie on the back, with the abdominal muscles relaxed. The physician, standing on the r. of the patient, should place his l. hand beneath the patient's back, close under the ribs, just external to the quadratus lumborum. The r. hand is laid flat over the anterior surface of the abdomen, in the mid-clavicular line, with the fingers pointing upwards, just below the liver. Pressure backwards, as if to meet the l. hand, is made by the r. hand. The patient should then be asked to draw a deep breath, and as he does so the rounded lower edge of the kidney is felt to slip between the opposing hands. When the ligaments of the kidney are relaxed—*mobile kidney*—the fingers of the r. hand may be able to palpate the upper border of the organ, and to retain it during expiration. A kidney is said to be "*floating*," when it can not only be readily palpated, but can be pushed below the umbilicus or freely moved about in the abdominal cavity.

**Percussion** does not enable us to define the margins of the kidney, for the organ is too deeply seated. The fact of primary importance in this connection is its relation to the colon, which, as just mentioned, is pushed forward by enlargements or tumours of the kidney. Consequently the anterior surface of such growths is always resonant, there being dulness at the side which is continuous with that at the back; whereas with enlargements of the spleen or gall-bladder there is dulness anteriorly, and resonance at the side.

#### PART C. URINARY DISORDERS, THEIR DIAGNOSIS, PROGNOSIS, AND TREATMENT.

§ 295. Routine procedure and classification. *Firstly*, having ascertained that the patient's LEADING SYMPTOM refers to the urinary apparatus; and, *secondly*, the data of his ILLNESS, particularly as to whether it is of an ACUTE, or (more probably in renal disease) CHRONIC nature; *thirdly*, we proceed to examine the urine. The ROUTINE EXAMINATION of the URINE in everyday practice consists of Inspection, Reaction, Specific gravity, Tests for Albumen and for Sugar. The subsequent and more detailed examination depends upon circumstances. As above stated, the examination of the urine stands in relation to renal disease, as the local signs do to diseases of other organs. There are very few diseases, certainly no common disorders of the kidneys, which are not attended by some change in the urine. On the other hand, the LOCAL EXAMINATION of the kidney, by palpation and percussion (§ 294), is difficult and relatively much less certain and instructive. On this account it comes last in our scheme of

examination, but it should never be omitted in any case which is the least obscure.

**Classification.** We will deal with urinary disorders under their respective cardinal symptoms as follows :—

Albuminuria . . . . .	§ 296
Hæmaturia . . . . .	§ 301
Pyuria . . . . .	§ 304
Alterations in the specific gravity . . . . .	§ 307
Polyuria . . . . .	§ 308
Glycosuria . . . . .	§ 309
Retention of urine . . . . .	§ 312
Suppression of urine . . . . .	§ 313
Incontinence of urine . . . . .	§ 314
Clinical significance of various deposits . . . . .	§ 315
Renal Enlargements . . . . .	§ 316

§ 296. **Albuminuria.** The numerous morbid conditions which may give rise to albuminuria, may be divided into three great anatomical (and clinical) groups: A. *acute inflammation* of the epithelium (acute nephritis or acute Bright's disease); B. *chronic inflammations and degenerations*; C. *renal congestions*, either active or passive, which include many cases of albuminuria independent of structural disease of the kidney, and which are mostly chronic in their course.

If, therefore, the illness came on recently, and is of an **Acute** character, turn first to § 296a (Acute Nephritis), and then to § 300 (Renal Congestions).

If, on the other hand, the illness is of some duration, and evidently of a **Chronic** kind, turn first to § 297 (Chronic Tubal Nephritis), and then the succeeding sections.

When the albumen is in small quantity, and there is also BLOOD or PUS in the urine turn to § 301 (Hæmaturia), or § 304 (Pyuria), respectively.

*The illness came on recently, and is of an acute order; the urine is diminished and contains a considerable quantity of ALBUMEN and TUBE-CASTS; it is or has been "SMOKY," from the presence of blood; ANASARCA is present; and there is a tendency to uræmia—the disease is ACUTE NEPHRITIS.*

§ 296a. **Acute Nephritis** (Acute Bright's Disease). In this disease the inflammation begins and predominates in the epithelium or parenchyma of the organ. The condition usually lasts

from five or six weeks, and may terminate in recovery or pass on to a chronic condition.

*Symptoms.* (1) The albumen is often in considerable quantity, amounting even to a quarter or a third of the urine in the test-tube, after the heat test. (2) The other characters of the urine are—(i.) It is scanty, sometimes only 10 or 20 ounces a day, or less. Consequently, the specific gravity is high, although the diurnal quantity of urea is diminished. (ii.) It is turbid or “smoky” from the presence of blood in the early stages. (iii.) Epithelial, granular, and blood casts, free renal epithelium, and red blood corpuscles are present. (3) Dropsy is general from the commencement, although it is first noticed in the face in the loose areolar tissue below the eyes and in the genitals. There may also be collections of dropsical fluid in the serous cavities. (4) There is a waxy pallor of the skin. (5) A degree of malaise, with discomfort and even pain in the loins, may be present, but there is only a slight elevation of temperature for about four or five days. (6) Uræmic symptoms may come on early, *e.g.*, (i.) occasional vomiting; (ii.) headache; (iii.) drowsiness. (7) If the disease goes on for any time the pulse becomes of high tension, and the second aortic sound is accentuated.

*Causes.* Acute nephritis is only very rarely a primary malady. (1) 90 per cent. of the cases supervene on an acute specific fever, and by far the most common of these is scarlet fever. (2) Chill, especially a sudden chill when the skin is perspiring. (3) Traumatism—*i.e.*, a blow on the kidney is an occasional cause. (4) The persistent use of certain drugs, such as cantharides and turpentine. (5) Inflammation secondary to disease of the urinary tract below the kidney (see Ascending Pyelo-nephritis). (6) Pregnancy is a marked predisposing, and sometimes exciting, cause.

*Prognosis.* Acute Nephritis may terminate in (1) complete recovery, the usual result when the treatment and hygienic surroundings are good. (2) Partial recovery. If the disease lasts longer than three months it usually develops into the condition known as large white kidney (chronic parenchymatous nephritis, § 297). (3) Death may occur from uræmia, from dropsy into the serous cavities, or from other complications. The chief *complications* are: (a) inflammations of the *serous* membranes, such as

pleurisy, pericarditis, or peritonitis, which are mostly latent—*i.e.*, attended by little or no pain; and (b) inflammations of the *mucons* membranes, such as bronchitis, gastritis, enteritis (causing diarrhœa), œdema of the lungs or of the glottis. Erysipelas, cellulitis, and various other *skin diseases* are very prone to attack patients with acute nephritis.

The prognosis, therefore, of acute nephritis is grave in proportion to (i.) the diminution of urine; (ii.) the development of uræmic symptoms; (iii.) the amount of dropsy present; and (iv.) the nature and severity of the complications.

*Treatment.* The indications are—to relieve the kidney by giving only bland non-irritating food, *e.g.*, milk; to increase the action of the skin and bowels; and to lessen local congestion. (1) To obviate the great liability there is to chill, the patient should be kept in bed; and for the same reason, all cases of scarlet fever should be kept in bed during convalescence, because they are so apt to develop this disease. (2) Diaphoretics, such as liquor ammon., acetat., antimon. tart., jaborandi or pilocarpine, warm baths, wet packs, hot-air baths. This treatment may be applied by means of a wicker cage placed upon the bed, and connected with a spirit lamp through an iron chimney at the foot. (3) Purgatives, such as pulv. jalap co. (30 grs. to 3j), are indicated. Saline purgatives are especially useful when there is much dropsy. (4) There is some difference of opinion about diuretics; some say that they irritate the kidney, others that they relieve the symptoms, and especially the dropsy. It is agreed that during the acute stage copious libations of water, but no other diuretic, should be used. Saline diuretics employed are pot., bicarb., cit., acet., and bitart. Scoparium and digitalis are given with caution, if the heart is feeble. (5) Local depletion by wet or dry cupping is especially indicated when the urine contains much blood. Counter-irritation over the kidneys, with poultices or leeches, has a similar effect. (6) Tonics must be given during *convalescence*, especially iron. An admirable prescription is liq. ferri perchlor., ℥ 15; liq. ammon. acet., 3j; ac. acet., ℥ 5 (to prevent decomposition). Animal food should be forbidden so long as albuminuria continues. In the treatment of renal disease three drugs are contra-indicated—opium, cantharides,



and turpentine. Mercury is generally added to these, but I have never seen any harm arise from its administration. For the treatment of Uræmia see § 298.

**Chronic albuminuria.** There are three anatomical varieties of chronic renal disease attended with more or less albuminuria, which, when occurring in their typical forms, present well-marked clinical distinctions, as shown in a tabular form below. In Chronic Tubal Nephritis (including large white kidney), the renal *epithelium* is primarily, and throughout the disease chiefly involved. In Chronic Interstitial Nephritis (Gouty Kidney) the *interstitial tissue* shows evidence of increase, and throughout the disease this is the most marked change; the arteries also, however, show hyperplasia of their middle coat.<sup>1</sup> This arterial change also occurs throughout the body, and is attended by a corresponding hypertrophy of the left ventricle. In the Amyloid (or Waxy) Kidney the *vessels* are primarily involved, the lardaceous degeneration beginning in the middle coat. Pathologists make many sub-divisions, but these represent the three clinically recognisable groups of chronic renal changes attended by albuminuria.

TABLE XXII.—Dr. Murchison's Table of Chronic Albuminuria.

	Quantity of Albumen.	Tendency to Uræmia.	Quantity of Urine.	Tendency to Dropsy.
Chronic Tubal Nephritis.	Large.	Moderate.	Diminished or normal.	Great.
Gouty Kidney.	Very small.	Great.	Increased.	Very slight.
Waxy Kidney.	Very great.	Slight.	Greatly increased.	Slight.

If the albumen is CONSIDERABLE, turn to Chronic Tubal Nephritis, Amyloid Kidney, or Chronic Renal Congestions. If there is only a TRACE of albumen, and the urinary signs appear to be slight in proportion to the debility and other symptoms, turn to Chronic Interstitial Nephritis.

*The illness is chronic, and the general symptoms of renal disease pronounced; generalised DROPSY is marked; the urine is scanty, and ALBUMEN and CASTS are abundant—the disease is CHRONIC PARENCHYMATOUS NEPHRITIS.*

§ 297. **Chronic Tubal Nephritis** (syns. large white or pale kidney, chronic parenchymatous nephritis, chronic desquamative or catarrhal nephritis, fatty kidney) may follow on acute nephritis,

<sup>1</sup> Cf. Introduction to this chapter.

or may develop insidiously. In the latter stages the connective tissue is increased, and if the patient lives long enough the kidney becomes a *Contracted Fatty Kidney*, or *Small White Kidney*.

*Symptoms.* (1) The albuminuria is considerable,  $\frac{1}{4}$  to  $\frac{1}{3}$  on standing; (2) the other characters of the urine are—(i.) the diurnal quantity is slightly diminished at first, but towards the end, when the kidney contracts, the quantity may be greater than normal; (ii.) the sp. gr. is not much altered in the early stages, but the urea is deficient throughout; (iii.) it is turbid, often with lithates; and recurrent hæmaturia may occur, especially if the condition has followed acute nephritis; (iv.) all forms of casts are met with (§ 292). (3) There is generalised dropsy, but most marked in the face. It may disappear towards the end, when the diurnal quantity of urine increases. (4) There are pallor, emaciation, weakness, and digestive disorder; and (5) cardio-vascular symptoms (§ 270) ensue.

*Etiology.* (1) Chronic tubal nephritis frequently follows acute nephritis, or (2) it may result from prolonged mechanical congestion of the kidney (as in cardiac disease, pregnancy, etc.). (3) Sometimes it comes on insidiously, without apparent cause. (4) Alcohol in excess predisposes.

*Diagnosis.* When the insidious form occurs in young women it is often mistaken for *anæmia*. In all cases of anæmia resisting iron, examine the urine for albumen. In the later stages it may be mistaken for *chronic interstitial nephritis*; but in that disease the patient is usually older, and see Table XXII. In certain cases which present *both renal and cardiac* symptoms it may be very difficult to say *which condition is the primary* one. In such cases it is important to note the following points:—(i.) If there is a *history* of rheumatic fever and previous attacks of dropsy, it is probable that the cardiac condition is primary. (ii.) If *other than mitral* systolic murmurs are present it points to cardiac disease; a mitral regurgitation murmur *alone* might be due to the cardiac failure following renal disease. (iii.) The *urine*, when there is any difficulty in diagnosis, is in both cases scanty and albuminous. Many tube-casts point to renal disease; the rapid clearing up of the urine after a short period of rest in bed points to heart disease. (iv.) A *hard pulse* favours kidney disease, but an irregular

soft pulse is found with cardiac failure secondary both to renal and to cardiac disease.

*Prognosis.* When once established the disease can never be cured, and even with careful diet and treatment the patient rarely lives more than a few (2 to 5) years. Death occurs as a consequence of dropsy, uræmia, or complications (as in acute nephritis). The prognosis is grave in proportion to (1) the amount of dropsy and albuminuria; (2) the diminution of urine; and (3) the presence of uræmic symptoms. If the patient survive for several years the prognosis improves; because, when the stage of contraction sets in life may, with care, be somewhat prolonged.

The *treatment* is much the same as that of acute nephritis (*q.v.*); but two points demand constant attention: (1) the avoidance of chill, by the wearing of flannel and resort to equable climates; (2) careful dieting, with the object of reducing the nitrogenous intake to a minimum. The latter is best accomplished by making the patient live entirely on milk, two to four pints per diem, and as much fluid as the patient can drink. If meat be taken, it should not exceed two or three ounces once a day. Stimulants, meat extracts, and animal soups should be avoided, unless evidences of a fatty heart are present. Purgatives should be administered so that the bowels act twice a day. Tonics, and especially iron, are the best drugs. The best tonic is that prescribed for convalescent acute nephritis (*q.v.*); it may be combined with a diuretic.

*The patient complains of lassitude, and other symptoms of* INCIPIENT URÆMIA *mentioned in § 273. There are only* TRACES OF ALBUMEN, *the diurnal quantity of urine is increased, dropsy is absent—the disease is probably* CHRONIC INTERSTITIAL NEPHRITIS.

§ 298. **Chronic Interstitial Nephritis.** (Syns. Contracted, Granular, or Gouty Kidney; Cirrhotic Kidney; Renal Fibrosis; Small Red Kidney; Chronic Non-desquamative Nephritis.) When “chronic Bright’s disease” is mentioned it is generally this variety of chronic renal disease which is referred to. It is accompanied by widespread cardio-vascular changes, as mentioned in the introduction to this chapter.

*Symptoms.* (1) The albuminuria in this disease is small in amount, and many samples of the urine may be examined without

finding any. In cold weather, however, when there is deficient skin action, there is generally a trace, especially after a chill or any cause which produces renal congestion. The other characters of the urine are:—(ii.) The diurnal quantity is greatly increased (may be to 100 ozs.). The patient often consults us because he has to get up at night several times to pass water. (iii.) The specific gravity is very low (1005—1012), owing partly to the deficiency in urea, but chiefly to the increased quantity of urine. The deficiency in total urea is not very great, but it should always be carefully estimated (§ 282). (iv.) The urine is clear, pale, and contains but few casts, and these are chiefly hyaline or granular (Fig. 79). (2) Dropsy is usually absent. If dropsy occur it is due to (i.) secondary cardiac failure, or (ii.) the supervention of acute nephritis. Sir George Johnson found a history of dropsy in only fourteen out of thirty-three cases. (3) The patient may look robust, but sometimes he has a greyish pallor. (4) The pulse indicates persistent high tension, and is often associated with hypertrophy of the left ventricle,<sup>1</sup> an accentuated aortic second sound, sometimes with a systolic apical murmur (probably regurgitant), and always sooner or later with a thickened condition of all the arterics. Later the heart may dilate, with consequent dropsy and albuminuria, and it may be hard to diagnose whether the kidney or the heart condition is primary or secondary (§ 297, *diagnosis*). (5) There is throughout a condition of chronic or incipient uræmia (§ 273) due to the deficient nitrogenous metamorphosis in the body, and the retention in the blood and tissues of the antecedents of urea, owing to deficient renal function.<sup>2</sup> These symptoms are indefinite, but in order of importance they are:—(i.) Insomnia and headache, symptoms which, occurring in the aged, should always lead us to suspect granular kidney; (ii.) gradual impairment of the mental and bodily vigour; (iii.) tremors and twitching of the muscles; (iv.) digestive disorders; (v.) dyspnœa, often paroxysmal.

<sup>1</sup> Chronic renal disease is often attended by hypertrophy of the l. ventricle, which is variously explained:—(i.) By the continuous high blood-pressure often present in such cases; or (ii.) By a spasm and thickening of the walls of the microscopic arterioles, which Sir George Johnson proved to exist in most cases of chronic renal disease, and which he regarded as due to the poisoned blood. (*Med. Chir. Trans.*, 1850—1873.)

<sup>2</sup> If pot. iod. be given (gr. xxx) iodine appears in the urine excreted by a healthy kidney in one hour. (Test by adding  $\text{HNO}_3$  to the urine, and starch—a blue colour develops.) But in granular kidney iodine is not found, or only faint traces.



*Course and Complications.* Apart from the existence of slight and intermittent albuminuria and persistent *high arterial tension*, non-urinary symptoms are the earliest, and often for prolonged periods the only evidences of this disease. In many cases the high arterial tension first reveals the disease to the physician; in other cases it is the *ophthalmoscopic changes* (renal retinitis, § 270), changes which may or may not be attended by failure of vision. Apart from the progressive enfeeblement, the disease is mostly revealed to the patient by the occurrence of one of its numerous complications. The most frequent and most serious of these is *cerebral hæmorrhage*, resulting from the prolonged high tension and consequent arterial degeneration. *Hæmorrhages* of various kinds may occur in other directions, such as epistaxis, or melæna. Epistaxis constitutes a kind of safety-valve, relieving the vascular system from more serious internal hæmorrhages, consequently it should not be checked. The *mucous membranes* are often affected, and intractable bronchitis or gastro-enteritis in an elderly person may be the condition which brings the patient under our notice; the *serous membranes* less often, though a latent form of pleurisy or pericarditis is not uncommon. *Skin diseases* are often very troublesome. The earliest symptom noticed in many cases is the itching of the skin. In the earlier stages urticaria and eczema are apt to occur; and in the later, erythematous, bulbous, desquamative, and hæmorrhagic eruptions.<sup>1</sup> The patient is liable, on exposure to cold, to attacks of *congestion of the kidney*, when the albuminuria and all the other symptoms are aggravated.

The *Diagnosis* from other forms of chronic renal disease is given in Table XXII., p. 501. This disease is also distinguished from *chronic parenchymatous nephritis* by the fact that the latter occurs in younger people. However, the diagnosis of this form of chronic renal disease from the other conditions which give rise to lassitude and DEBILITY is often a question of much greater difficulty, and this will be fully dealt with in Chapter XVI.

*Etiology.* (i.) I have assisted at an autopsy in a well-marked case of granular kidney in a child of nine<sup>2</sup>; but the disease almost

<sup>1</sup> See also Thursfield, Med. Chir. Trans., 1901.

<sup>2</sup> In childhood the leading symptoms are general pigmentation, polyuria, and headache. The first is explained by the possible invasion of the adrenals. The headache is often worst on rising in the morning, *i.e.*, like a high tension headache.

invariably occurs in *persons of middle age or advanced life*. Out of 376 cases admitted into the Paddington Infirmary 317 were over 40 years of age; 251 were over 50, and 203 over 60. (ii.) *Gout* and a gouty habit of body is the most important causal factor. In many cases there is a long history of persistent lithuria, and in a large proportion of cases of granular kidney the joints and ears show evidences of gouty deposit, hence the name "gouty kidney." (iii.) An *indolent* life, and (iv.) *Chronic lead poisoning* are undoubtedly causes, not only of gout, but of granular kidney.

*Prognosis.* The course of the disease, as already mentioned, is prolonged. With care and attention to diet the patient may live for five, ten, or more years, but the disease can never be cured. The amount of albumen is no criterion as regards prognosis in chronic interstitial, as it is in chronic parenchymatous, nephritis. The prognosis is grave in proportion (1) to the duration of the disease; (2) to the evidences and degree of uræmia present; (3) the degree of cardiac failure; and (4) the presence and severity of the complications (*vide supra*). Life is most frequently terminated by cerebral hæmorrhage or some other complication. But a large number of these cases die of acute uræmia (§ 273), as the records of the Paddington Infirmary show. Older authors described this as death by "serous apoplexy," thinking that the serum which replaced the atrophy of the brain was the cause of pressure upon that organ.

*Treatment.* Diet is the point of chief importance. The amount of proteids should be reduced to an absolute minimum, and alcohol forbidden. All chances of chill should be avoided by clothing in flannel, and living in equable climates; and the action of the skin should be maintained. The arterial tension should be reduced (§ 64); this takes the strain off the heart, and will cure symptoms such as headache and insomnia due to high pressure. Purgatives, such as *mist. alb.*, will do a great deal for this, at the same time enabling the bowels to drain off the poisonous substances which ought to be eliminated by the kidneys. Tonics are useful, such as *nux vomica*, and even *digitalis* for heart failure. The action of the latter must be carefully watched (special heed being given to the pulse), lest it produce apoplexy or other hæmorrhages. Iron is not of much use, and may do harm by leading to

constipation. *Symptomatic treatment.* (i.) Liq. trinit.  $\text{m}\text{j}$ , b.d. will cure headache when due to high tension. (ii.) Pot. iod. relieves tension, and may prevent further arterio-sclerosis. (iii.) For the attacks of "renal asthma," amyl nitrite, chloroform, or venesection may be necessary. (iv.) For restlessness, chloral and bromides are good.

The treatment of this disease very often resolves itself into *the treatment of uræmia*, which is as follows, the indications being—1, to eliminate the poison as rapidly as possible; and 2, to alleviate the symptoms. In *chronic* uræmia a daily dose of Epsom salts may be taken; the skin must be encouraged to act; diuretics such as pot. bicarb. and acet., sp. æth. nit. and scoparium administered; and digitalis if the heart is dilated. Large quantities of water should be taken. To relieve tension and headache nitroglycerine is valuable (see also § 64).

For *acute* uræmia—muttering delirium, convulsions, coma (diagnosis of uræmic coma, see Chapter XIX.)—a brisk hydragogue purgative must be given at once; such as pulv. elat. co., pulv. jalap. co., or a concentrated solution of mag. sulph. The skin must be made to act by means of hot packs, hot air or vapour baths, or pilocarpine (gr.  $\frac{1}{8}$  to  $\frac{1}{4}$  hypodermically). Venesection (10 to 20 ozs.) did a great deal of good in many of my infirmary cases, and undoubtedly averted a fatal issue. Transfusion of normal saline solution (0.75 per cent. NaCl) compensates for the loss of fluid by bleeding or purgation, and may with advantage be adopted after venesection. Chloroform relieves the convulsions.

*There is abundant albumen with the passage of LARGE QUANTITIES of urine, but little tendency to dropsy and uræmia; the patient has a history of prolonged SUPPURATION, or of SYPHILIS; and there may be evidences of lardaceous disease elsewhere—the disease is LARDACEOUS KIDNEY.*

§ 299. **Amyloid Kidney** (Waxy or Lardaceous Kidney) is generally part of a widespread lardaceous disease involving the liver (enlargement), spleen (enlargement), and intestines (diarrhœa).

*Symptoms.* The albumen, though it may be small in quantity in the early stage, is very abundant, amounting to three-fourths or more, when the condition is established. The other characters

of the urine are :—(i.) the diurnal quantity is greatly increased even to 150 ozs. ; (ii.) the specific gravity is very low, but the urea is not diminished till the latter stages ; (iii.) the colour is pale and clear ; (iv.) all varieties of casts may be found, including hyaline and fatty casts. (2) There is great pallor of the surface and anæmia ; but there may be no dropsy till quite the end of the disease. In cases with great cachexia dropsy *may* occur early. (3) Evidence of lardaceous disease in other organs is present—liver, spleen, and intestines, consequently hæmorrhages may occur from different parts. The amyloid disease of the bowel gives rise to very intractable diarrhœa, a symptom which often accompanies amyloid kidney.

It is important for the *diagnosis* to ascertain the history of a *cause*, namely, (a) prolonged suppuration, either from a chronic abscess, chronic phthisis, or caries. Dr. Murchison used to be of the opinion that caries of the vertebræ, even without definite formation of an abscess, could give rise to lardaceous disease of the viscera, especially the kidney. (b) Constitutional syphilis is the second of the two great causes which bring about lardaceous disease.

*Prognosis.* The course of the disease is protracted. The patient may live for several years, dying by exhaustion from diarrhœa, or other complications ; very rarely from uræmia due to the super-vention of acute nephritis. With careful treatment patients may live for many years, or even recover if the disease is seen in a very early stage ; but the prognosis is bad in proportion to (1) the amount of albuminuria, and (2) the extent of the involvement of the other organs.

*Treatment.* Alkalies have been reputed not only to prevent, but also to relieve the lardaceous process, *e.g.*, liquor potassæ (5 minims) ; the tartrates and citrates of the alkalies are also administered. Iodine, especially in the form of iodide of potassium or iodide of iron, particularly when syphilis is in operation, should be given. The most troublesome complication is diarrhœa. The only remedies which in my experience are of any use are liq. ferri pernit. (15 minims) ; or pil. plumbi cum opio (5 gr.) continued every four hours until the diarrhœa ceases. Opium may be administered in this form of renal disease, when there is no tendency to uræmia. The *preventive treatment* of



lardaceous disease consists in the adequate treatment of syphilis in its early stages; and in curing prolonged suppuration, especially when this occurs with chronic profusely discharging ulcers of the leg.

§ 300. In **Renal Congestion** (secondary albuminuria) there is sometimes a very considerable amount of albumen in the urine; but the urinary and other symptoms do not conform to the foregoing types. Casts are mostly absent, never abundant, and the constitutional disturbance, apart from the *primary* malady, is slight.

*If the albuminuria is MARKED and CONSTANT, and especially if the urinary symptoms are associated with symptoms referable to some other organ, it is probably PASSIVE renal congestion due to* I. CARDIAC DISEASE; II. ASCITES or ABDOMINAL TUMOURS; or III. PREGNANCY.<sup>1</sup>

*If the albumen is SLIGHT in amount, and especially if it be TRANSIENT, it is probably ACTIVE renal congestion due to* IV. CHILL TO THE SURFACE; V. PYREXIA or other BLOOD DISORDER; VI. DRUGS; VII. DYSPEPSIA or HEPATIC DERANGEMENT; VIII. DERANGED INNERVATION; or IX. FUNCTIONAL ALBUMINURIA.

I. CARDIAC DISEASE (the cardiac kidney)<sup>2</sup> is the most frequent of the above-mentioned causes of albuminuria. Albuminuria is a very common accompaniment of mitral valvular disease, and of the dilated r. heart which so frequently follows chronic bronchitis and emphysema. At first the kidney is only congested, but in the later stages the epithelium may become affected and the interstitial tissue increased. The diagnostic features of the albuminuria in such cases are:—1. The amount of the albumen is always considerable, and may be very great. 2. The urine is scanty, high-coloured, of high specific gravity, and there may be blood cells, renal cells, or even casts; nevertheless these latter may disappear when the cardiac condition is relieved. 3. There are evidences of the cardiac disease which has produced the renal

<sup>1</sup> In the *later* stage of pregnancy passive congestion by pressure undoubtedly occurs, but *throughout* there are evidences that the renal mischief is due to a toxæmia, and therefore due also to congestion of an active kind.

<sup>2</sup> It is well to bear in mind that when both cardiac and renal disease are present they may be associated in three ways:—(a) Cardiac disease may produce renal disease in one of the above-mentioned ways. (b) Renal disease may produce cardiac disease, as when acute nephritis or granular kidney lead to cardiac hypertrophy and failure. (c) They may both be the result of a common cause, *e.g.*, gout!

disease. In some cases it is difficult to decide which of these was primary, as above mentioned (§ 297, *diagnosis*).

Cardiac disease may give rise to renal disease in three ways:—(i.) In the manner just stated. (ii.) *Embolism of the kidney* is one of the consequences of endocarditis (acute or chronic). In this condition the albuminuria appears suddenly with hæmaturia and constitutional symptoms, and disappears equally suddenly in a few days. (iii.) Some cases of *aortic valvular disease* (the mitral being healthy) have been attended by temporary albuminuria. The explanation is not quite obvious. In these cases the compensatory hypertrophy and dilatation were great, and the arterial tension high; and it seems probable therefore that the albuminuria may have been due to an *active* renal congestion.

II. ASCITES and ABDOMINAL TUMOURS. Here the albuminuria is due to pressure on the renal veins. This condition is recognised by—(1) The amount of albumen is generally moderate; (2) There is abdominal enlargement with the signs of fluid or tumour; (3) The albuminuria will disappear on removing the cause. There are two fallacies to be remembered before diagnosing albuminuria as due to ascites:—(i.) both albuminuria and ascites may be the product of some common cause, *e.g.*, heart disease; and (ii.) the ascites may be the result of a general dropsy due to renal disease.

III. PREGNANCY is an undoubted cause of albuminuria, and according to Playfair<sup>1</sup> it occurs in 20 per cent. of parturient women after the third month. It also seems certain that permanent and ineradicable renal disease may, in some cases, date from pregnancy. According to some, the albuminuria of pregnancy is due to pressure on the renal veins; a view that is supported by its more frequent occurrence in primiparæ, in whom the abdominal walls are more rigid. But, on the other hand, the albuminuria may occur before the uterus is large enough to cause pressure on the renal veins. These and other considerations point to the conclusion that it is probably due to some blood change associated with the parturient state. The clinical features are:—(1) the amount of albumen is not usually great, and the urine is otherwise normal or very much as in I., *supra*. (2) Ophthalmoscopic changes (§ 270) may be present; but (3) these and the urinary symptoms disappear within 2 or 3 weeks of labour unless permanent renal disease has been induced. The treatment is discussed below.

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<sup>1</sup> "Handbook of Midwifery," 2nd edition, vol. i., p. 222.

The remaining causes of albuminuria are probably due to ACTIVE congestion of the kidney—

IV. CHILL TO THE SURFACE. Chill to the surface may result in albuminuria, but in such cases the kidney is rarely quite healthy. This condition is recognised by:—(1) The amount of albuminuria is never very great, and it does not last for more than a few days; (2) the urine is otherwise normal, or may deposit lithates; (3) the patient, in other respects, is healthy, or complains only of slight bronchial catarrh or coryza.

V. PYREXIA and other BLOOD DISORDERS. This cause of albuminuria is characterised by:—(1) An elevated temperature; in hyperpyrexia albuminuria is invariably present. (2) Tube-casts are absent unless there be active renal disease. (3) Other evidences of the toxic blood state are present, namely:—(i.) Various acute specific fevers, *e.g.*, diphtheria, where albuminuria may be present without high temperature. In scarlet fever albuminuria frequently comes on between the 16th and 26th day, at which time also acute nephritis may supervene; and, to avoid this risk, scarlet fever patients should be kept in bed three to four weeks. Transient albuminuria may occur in secondary syphilis, between the 6th and 8th weeks of the disease. If albuminuria occurs in the later stages of the disease, it may be due to lardaceous disease or gumma of the kidney. (ii.) Acute pneumonia is sometimes, and (iii.) Acute gout is very frequently, accompanied by albuminuria. (iv.) Albuminuria may also occur in diabetes (in which it is a grave sign), in pernicious anæmia, leukæmia, and the reaction stage of cholera.

VI. Various DRUGS, such as morphia, quinine, phosphorus, arsenic, cantharides, cubebs, copaiba, turpentine, salicylic acid, mercury, and carbolic acid, may give rise to albuminuria. This cause is recognised by (i.) the presence of the drug in the urine; (ii.) there may be a history of the administration of the drug; and (iii.) the albuminuria disappears when the drug is stopped.

VII. DYSPEPSIA and LIVER DERANGEMENT are sometimes accompanied by albuminuria. The symptoms of hepatic congestion may be present, showing the intimate connection between the hepatic and renal functions (§ 240). Albuminuria is sometimes present with that form of dyspepsia which is accompanied by oxaluria. Certain articles of diet have been known to be attended by albuminuria. Thus, cases have been recorded in which albuminuria followed the ingestion of shell-fish, eggs in excess, cheese and alcoholic bouts. In many of these instances the condition is probably albuminosis (§ 288).

VIII. DERANGED INNERVATION may be attended by albumen in the urine, such as (1) burns and other causes of severe shock. (2) In exophthalmic goitre the albuminuria is usually a temporary condition, though it may last for months. It may vary in amount at different times of the same day, a fact which tends to show that it is of vasomotor origin. The urine in other respects is healthy. (3) Excessive study or other cause of nerve strain has been reported to have occasioned albuminuria. (4) Certain cases of cerebral tumour, and other conditions in which there is increased intracranial pressure, have been attended by albuminuria.

IX. PHYSIOLOGICAL or FUNCTIONAL Albuminuria. A *cyclic* form of albuminuria, or the "albuminuria of adolescence," has been described,

It appears regularly at some time each day, usually in the morning or after a cold bath. It is usually absent at night, or when the patient retains the horizontal position, and is possibly therefore of vasomotor origin. So also is a form of *paroxysmal* albuminuria which has been described, and which is probably closely related to paroxysmal hæmoglobinuria (below). It appears at intervals, without any apparent cause, and lasts for a few days or weeks at a time. Some of the reported cases were probably early stages of Raynaud's disease, others were perhaps associated with oxaluria.

The *prognosis* of albuminuria due to congestion is very much that of its cause. Before giving a prognosis it is important to thoroughly and repeatedly examine the urine, for casts in particular, so as to be satisfied that the kidneys are structurally healthy. When due to prolonged dyspepsia and liver derangement, interstitial nephritis may supervene in time, if the diet is not properly modified.

*Treatment.* In most of the conditions the treatment must be directed to the causal agent. Rest in bed will do a good deal for the renal complication of cardiac disease. In the *albuminuria of pregnancy* careful investigations should be made, and the amount of urea watched. If, (1) there is a clear history of renal disease prior to pregnancy, or (2) puerperal eclampsia has occurred in previous pregnancies, or (3) the renal disease, no matter of what kind it may be, is distinctly *progressive in its nature*, then premature labour should be induced. For the treatment of cyclic and paroxysmal albuminuria general hygienic and dietetic rules must be followed.

§ 301. **Hæmaturia.** When the patient is "passing blood" in the urine, an endeavour should be made to ascertain if the blood comes chiefly at the beginning of micturition, chiefly at the end, or whether it is intimately mixed with the urine and gives to it a "smoky" tint. For the tests for blood in the urine see § 285. The fallacies of menstrual blood, and certain drugs which give a red colour to the urine, must be avoided by using a catheter.

*A. If the blood is bright crimson and comes chiefly AT THE COMMENCEMENT of micturition, it is probably of URETHRAL or PROSTATIC origin.*

In these circumstances, which are mainly of surgical interest, there will probably be a history of injury or gonorrhœa. In congestion or abscess of the prostate there are local pains and tenderness, and rectal irritation.

*B. If the blood comes most freely AT THE END of micturition, and especially if in clots, it is probably of VESICAL origin.*

The COMMONEST CAUSES of vesical hæmorrhage are :—

I. ACUTE CYSTITIS, chiefly at its onset (see § 305).

II. CALCULUS, or stone, in the bladder. Here the hæmorrhage is worse after exercise, moderate in amount, and there is pain, which, like the bleeding, is worse at the end of micturition and after exercise, and is



frequently referred to the point of the penis. The ensuing cystitis may complicate the symptoms and render the diagnosis of stone difficult, but its detection by the sound is conclusive.

III. TUMOURS of the bladder. The hæmorrhage here, especially in *villous* tumours, is usually excessive in amount. Shreds of the growth may be passed, and cystitis may develop. In *cancerous* tumours the hæmorrhage is more or less intermittent, and resists treatment; there are pain and cachexia, and sometimes the growth may be palpable above the pubes.

Some of the LESS COMMON CAUSES of vesical hæmaturia are TUBERCULAR DISEASE of the bladder (when the bacillus may always be found), VESICAL VARIX, certain constitutional diseases such as SCURVY and PURPURA, and BILHARZIA HÆMATOBIA.

BILHARZIA HÆMATOBIA, "Endemic Hæmaturia," occurs in Egypt and South Africa. It is due to the presence of a parasitic trematode. The adult worm is only found in the portal vein and its radicals, but the ova and liberated embryos migrate into and block the veins of the bladder, ureters, or kidney pelvis, causing hæmorrhage and great changes in their mucous surfaces. It is not known how the parasite enters the body, but it is believed to enter the rectum (some say the bladder) while bathing. This cause of hæmaturia may be readily detected by the presence of the highly characteristic ova in the urine (Fig. 90). They are equipolar ovoid bodies with a nipple-like projection at one end, and can be easily seen under a  $\frac{1}{2}$ -inch objective. Urotropin relieves the vesical symptoms, but the best treatment is large draughts of water containing benzoic acid (20 to 30 grs. daily), and every fourth or fifth day methylene blue (4 grs. t.d.).<sup>1</sup> The hæmorrhage may be very great and severe anæmia result; death generally takes place by pneumonia.

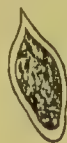


Fig. 90.—Egg of Bilharzia Hæmatobia, magnified about 100. Life history, see Table XIX., p. 388.

The *diagnosis* and *treatment* of these various vesical conditions (excepting the last-named) is mainly in the hands of the surgeon; but temporary relief generally attends rest and the administration of henbane.

*C. If the blood is INTIMATELY MIXED with the urine, causing it to assume a "smoky" tint, it is probably of RENAL origin. In these cases also the tests for blood should be carefully applied, and fallacies avoided (§ 285).*

The CAUSES of RENAL HÆMORRHAGE may for convenience be grouped into—inflammation (I.); calculus and other causes of pyelitis (II.—IV.); local conditions (V.—VII.); causes from distant parts (VIII.—XI.); paroxysmal hæmorrhage and parasites (XII.—XIII.).

I. In acute nephritis the blood usually gives rise to the characteristic "smoky" urine, and the deposit contains casts (§ 296a).

<sup>1</sup> Mr. P. S. Lebeau, R.A.M.C., Report of the Med. Soc. Lond. in *The Lancet*, May 10, 1902.

II. Renal calculus (see below).

III. Tubercular disease of the kidney (§ 306).

IV. Any of the other causes of pyelitis (§ 306) may give rise to red corpuscles in the urine in larger or smaller amounts.

V. Malignant and other tumours of the kidney cause profuse and sometimes intermittent hæmorrhage (§ 316).

VI. Villous disease of the pelvis of the kidney.

VII. Injury to the kidney (below).

VIII. Passive congestion of the kidney, for example in heart disease, or chill.

IX. Embolism of the kidney (see Endocarditis, § 42).

X. Blood poisons—fevers, scurvy, purpura, etc.

XI. Drugs, such as cantharides or turpentine.

XII. Paroxysmal hæmoglobinuria (below) differs from all the foregoing in the absence of blood discs, though blood colouring matter is plentiful in the urine.

XIII. Parasites, *e.g.*, Bilharzia Hamatobia (see above). *Filaria sanguinis hominis* usually causes chyluria, but hæmaturia also may occur.

§ 302. **Renal Calculus and Renal Colic.** Calculi may form either in the pelvis of the kidney, or more rarely, in its substance. Perhaps the commonest form consists of *uric acid* and urates mixed in varying proportions. These form stones of light brown colour, either round or branching (for tests see § 283). These are the commonest stones in subjects of the gouty diathesis, and those whose urine habitually deposits lithates. The other variety, which is dark brown in colour, consists of *oxalate of calcium*. This gives rise to much acuter symptoms, for they bristle with sharp pointed crystals which wound the mucosa. Calculi are often multiple. Compound stones consisting of an oxalate nucleus, or alternate layers, are met with. Phosphates and cystine are only very rarely met with in renal calculi. Various *events* may happen. 1. A calculus may remain in the renal pelvis, giving rise to chronic pyelitis (§ 306) for years; or 2, by its movement produce acute symptoms, **RENAL COLIC**. 3. It may obstruct the ureter and lead to hydro- or pyo-nephrosis (§ 316). 4. If the other kidney is not healthy sudden blocking may lead to obstructive suppression (§ 313). 5. It may pass into the bladder and result in cystitis. 6. Small stones may be voided through the urethra as “gravel.” 7. In rare cases small calculi become encysted and quiescent. The typical clinical history of renal calculus consists of (a) *attacks of renal colic*, separated by (b) *intervals* in which the symptoms are those of calculous pyelitis (§ 306).

The *symptoms of renal colic* consist of severe paroxysms of lancinating pain starting in one loin shooting down to the testicle or vulva on that side ; attended by vomiting, shivering, sweating, pallor, and a certain amount of collapse. These symptoms are in most cases followed by hæmaturia, the urine containing blood discs, and pus cells, but usually no casts. Crystals are also present, and guide us as to the nature of the stone. It is with the oxalate calculus that most blood and pain occur. The diagnosis of renal from other forms of colic is given in the form of a table (XV., p. 310). All the symptoms of renal colic may arise simply from the irritation of *fine crystals*. They may also be produced without alteration in the urine by *movable kidney* ; or by the passage of *clots* of blood, or caseous material (?tubercular) down the ureter. *Malignant* disease of the kidney may be mistaken for calculus, but in that case the blood is more copious and more constant, and the pain is less severe, but more continuous.

*Treatment*—(1) of the colic and (2) during the intervals. 1. The treatment of an attack of *renal colic* consists mainly in the relief of the symptoms—pain, vomiting, and collapse. Various forms of hypnotics may be tried (see neuralgia), but usually nothing avails except morphia, and it may safely be given unless there is reason to fear the renal parenchyma is also diseased. Effervescent citrate of potassium with sp. am. ar. may be administered with advantage, and rest should be enjoined. 2. The treatment in the intervals resolves itself into (i.) the prevention or solution of the stone, and (ii.) treatment directed to the pyelitis. (i.) Dietetic treatment is of great use in some cases. If uric acid is being passed, the treatment is the same as that for lithæmia (§ 251). If oxalates are being passed, any dyspepsia should be carefully treated ; such articles of diet as rhubarb, tomatoes, cabbages and onions, sweets and alcohol, should be avoided. The urine in all cases should be kept diluted by drinking plenty of fluid. The alkaline waters are very useful here, such as those of Vichy, Ems, and Contrexville. In uric acid calculus, large doses of alkaline salts are certainly useful, especially the citrate and the acid tartrate of potassium. Begin with 50 grs. of pot. cit. in 4 ozs. of water every four hours until the urine is alkaline, and

then give an effervescing drink, consisting of 1 drachm of sod. bicarb., and 40 grs. of citric acid in 4 ozs. of water, three times a day. This treatment should not be continued if the urine is or has become ammoniacal. (ii.) For the treatment of pyelitis see § 306.

**Injury of the Kidney**, laceration or rupture, is usually caused by a fall on the back or loin, or in "buffer-accidents" on the railway, during shunting operations. There may be no bruising or external signs, but a laceration of the kidney may be inferred from (1) the history of such an accident; (2) a tense swelling (due to extravasated blood) with increased area of dulness in the region of the kidney; and (3) copious hæmaturia. In a few cases there is no hæmaturia, and the other two evidences have to be relied on. Immediate operation is advised by Mr. Bland Sutton and others,<sup>1</sup> the collapse being treated by saline injections.

§ 303. In **Paroxsymal Hæmoglobinuria** (P. Hæmatinuria, Roberts) porter-coloured urine is passed at intervals. An attack commences abruptly with (1) *a rigor* or "chilliness," nausea and malaise; and (2) lumbar pain. (3) An hour or so later the patient passes dark highly albuminous urine, showing the spectroscopic band of methæmoglobin; containing no red discs, but a quantity of amorphous granular matter. It has a sp. gr. 1020—22, a slight excess of urea, and deposits crystals of oxalate of calcium. Each attack lasts a few hours, and passes off as suddenly as it came, but only to recur in a few hours' or few days' time. In the intervals the general health is fair, but later the patient becomes anæmic and languid. Relapses recur for months or years without fresh exposure to "chill."

The *causes* are obscure. The symptoms clearly indicate, as Sir William Roberts points out,<sup>2</sup> a sudden transitory congestion of the renal capillaries with transudation but *no rupture*. In 90 per cent. of the cases (Roberts) the attacks are connected with chill to the surface, and this seems to determine a reflex vaso-motor dilatation of the renal vessels. The disease is sometimes associated with rheumatism, oxaluria (the sharp crystals of which were thought by some to produce the attacks), malaria, mental or physical over-exertion, and dyspepsia.

The *treatment* consists of rest in bed during the attacks, with warmth; and hyoscyamus internally. Persons predisposed to such attacks should avoid exposure to cold, and take iron and quinine. In one case seen by the author bromide of ammonium had a marked effect in preventing the attacks.

SYMPTOMATIC HÆMOGLOBINURIA is a symptom which may occasionally accompany Raynaud's disease, malaria, severe burns, and acute infective diseases.

TOXIC HÆMOGLOBINURIA may be produced by toxic doses of chlorate of potassium, naphthol, pyrogallie acid, carbolic acid, arsenuretted hydrogen, and carbonic oxide.

<sup>1</sup> Discussion at the Clin. Soc., *Lancet*, Nov. 17, 1899.

<sup>2</sup> Reynolds' "System of Medicine."



*The patient complains of LASSITUDE and ill-health, which have come on gradually; the urine is found to contain PUS (§ 286) or pus corpuscles (§ 292), i.e., there is PYURIA. With few exceptions (see footnote, p. 518), when the pus comes from the BLADDER the urine is ALKALINE and the pus remains diffused through the urine; but when it comes from the KIDNEYS or any other part of the urinary passages the urine is ACID and the pus settles at the bottom. Pus cells produce a trace of albumen in the urine.*

§ 304. **Pyuria.** If we except the rupture of an abscess into the urinary passages,<sup>1</sup> there are three sources of pus in the urine,

A. From the **Urethra** (e.g., gonorrhœa).

B. From the **Bladder** (cystitis).

C. From the **Kidney** (pyelitis); of which there are three chief forms—**CALCULOUS P.**, **TUBERCULOUS P.**, and **ASCENDING P.**

It is believed by some observers, that persons in health may pass a few pus corpuscles (i.e., leucocytes); but it is extremely probable that these are always derived from the generative organs (male or female), and that the occurrence of *any* pus cells in a properly collected catheter specimen is always pathological.<sup>2</sup>

When the presence of pus is suspected, the reaction should be tested immediately after it is passed, before decomposition can set in. Decomposition makes the urine ammoniacal, and therefore alkaline.

**A.** *The pus comes chiefly at the BEGINNING OF MICTURITION and the urine is ACID*—it comes from the URETHRA, and may be caused, practically, by one of three conditions:—

I. **URETHRITIS.** There is pain, swelling, and redness of the meatus, scalding during micturition, and discharge of pus apart from micturition.

II. **PROSTATIC ABSCESS** is known by:—(1) pain at the end of micturition; (2) the finger in the rectum detects a tender, fluctuating swelling; (3) the symptoms closely resemble those of vesical calculus, with concurrent cystitis. It may be distinguished from this, however, by—(i.) a

<sup>1</sup> **Abscesses bursting into the urinary tract.** The abscesses most liable to burst into the urinary tract are—(a) prostatic abscess (above); (b) perineal abscess; (c) pelvic cellulitis; (d) psoas abscess; (e) perinephric abscess; and (f) abscess of the liver; and there are also many other sources. (i.) The urine is usually acid; (ii.) the pus is in large quantity and settles at the bottom; (iii.) there is a clinical history of abscess prior to the appearance of pus in the urine; and (iv.) localising signs of the abscess may be present.

<sup>2</sup> In some cases there is a history pointing to leucorrhœa or gleet, but the quickest way of settling this point is to draw off the urine by catheter.

history of gonorrhœa, which is the chief cause of prostatic abscess; (ii.) the signs on examination per rectum; and (iii.) a discharge occurring in the intervals between micturition.

III. PERINÆAL ABSCESS is detected by the local signs.

**B.** *The pus comes chiefly at the END OF MICTURITION or is intimately mixed with it, the urine is ALKALINE when tested immediately after it is passed*<sup>1</sup> (*alkaline pyuria*)—the pus comes from the BLADDER, and is indicative of CYSTITIS.

§ 305. **Cystitis**, or inflammation of the bladder, occurs in two well recognised forms—acute and chronic.

(a) In ACUTE CYSTITIS. (1) In this condition the pus is in small amount, and in severe cases there may be considerable hæmaturia at the outset. At first the urine is acid, but it soon becomes alkaline, and ropy with pus and mucus. (2) There are pain and tenderness in the hypogastrium. (3) Micturition is frequent and painful (“scalding”). After micturition the pain is relieved for a short time, unless the cystitis is due to stone in the bladder, when the pain is severe *after* micturition, because the inflamed walls of the emptied bladder then come into contact with the stone. (4) There is generally marked constitutional disturbance, with pyrexia.

(b) In CHRONIC CYSTITIS (which may supervene upon the acute form, or the cystitis may be chronic from the outset), there is (1) a larger amount of pus. (2) The urine is markedly alkaline, directly it is passed, and contains a large amount of ropy mucus. (3) The pain and other symptoms are less severe than in acute cystitis.

*Etiology.* (i.) Gonorrhœa causes the most severe and often fatal form of acute cystitis and pyelo-nephritis. Other causes are (ii.) stone or foreign bodies setting up irritation; (iii.) injury by instruments or foreign bodies introduced by the patient; (iv.) the use of catheters which have not been rendered thoroughly aseptic; (v.) cancer, villous disease, and other tumours of the bladder; (vi.) urine decomposing in the bladder, as in stricture urethræ, prostatic enlargement and other causes of retention of the urine (§ 312); (vii.) various nerve complaints, producing

<sup>1</sup> At the outset of acute cystitis the urine may be acid, and it may become acid again in the stage of recovery from chronic cystitis. It may also be acid in the early stage of tubercle and other new growths of the bladder. In all other conditions in which the urine contains pus derived from the bladder the reaction is alkaline.

paralysis and retention; (viii.) extension from a urethritis or inflammation from adjacent organs, as in pelvic cellulitis; (ix.) tubercle not infrequently affects the bladder, when bacilli are found in abundance; (x.) other microbes are now known to affect the bladder (compare iv. above), notably the bacillus coli communis, which produces a mild cystitis, and which is very apt to ascend (see infective pyelo-nephritis); (xi.) various constitutional states, such as gout, lithæmia, and diabetes are said to predispose to cystitis; (xii.) drugs, *e.g.*, cantharides or turpentine. The diagnosis of these causes is mainly accomplished by the surgeon.

*Differentiation.* 1. Cystitis due to VESICAL CALCULUS. In addition to the symptoms of simple cystitis there is (i.) pain at the end of micturition, lasting for some time after, very severe, shooting down the urethra; (ii.) hæmaturia is common, though in some cases it may be so slight that it is detected only by the microscope; (iii.) a history of renal colic (§ 302).

2. Cystitis due to NEW GROWTH IN THE BLADDER, or ULCERATION, is characterised by (i.) paroxysms of lancinating pain, quite independent of micturition and movement; (ii.) copious hæmorrhage at intervals, occurring without apparent cause; (iii.) the urine may contain cancer cells or tubercle bacilli; a tumour may be felt per rectum or through the abdominal wall. (iv.) Cystoscopic examination may settle the diagnosis.

*Prognosis.* Cystitis is not dangerous to life unless the inflammation spreads upwards from the bladder to the kidneys and produces pyelo-nephritis; but, on the other hand, it is a very troublesome, painful complaint, and has a special liability to recur. When the cause is not removable, *e.g.*, in cystitis due to tumours of the bladder, the prognosis is very grave. When it is due to retention of urine (such as that caused by the atony of the bladder in old age), and when it is due to gonorrhœa, it tends to cause ascending pyelitis and pyelo-nephritis. When there is pre-existing hydronephrosis (§ 316), and acute cystitis develops, the inflammation is almost certain to extend upwards to the kidney, and so lead to pyonephrosis.

*Treatment.* The cause must be sought for, and, if possible, removed. (a) In the *acute* form, absolute rest in bed with milk diet is necessary. Copious libations of water, barley-water and other bland fluids are called for. Alkalies allay the irritability of the bladder. Mild laxatives should be given, combined with hyoseyamus. Boric acid, 10 to 30 grs. thrice daily, in large draughts of water is valuable. Soothing drugs, *e.g.*, buchu, triticum repens,

and uva ursi are useful. Hot sitz-baths and morphia suppositories are given to relieve the pain. It is useful to administer internal antiseptics, such as quinine and salol, and a new remedy called urotropin<sup>1</sup> (7 grs. three times a day). (b) For the *chronic* and *subacute* forms:—wash out the bladder with hot water and boric acid. Sir Henry Thompson recommended that it is better to use a strong solution of boric acid, not exceeding 2 ozs. at each sitting, than to wash out with large quantities. Benzoic acid (gr. xv. t.d.s.) renders the urine acid.

**C.** *The pus is associated with a urine which is ACID when freshly passed (acid pyuria), the pus cells are at first disseminated through the urine, but in a short time they settle down as a SEDIMENT, and there are PAIN, and perhaps SWELLING, of the kidney—the pus comes from the kidney, and the disease is PYELITIS.*

§ 306. **Pyelitis**, or inflammation of the pelvis of the kidney, is indicated by the symptoms just mentioned. The urine, which is acid unless there be concurrent cystitis, contains in addition to pus cells (Fig. 82), epithelial cells from the renal mucosa; but, unless the renal parenchyma is involved, no casts and no albumen in excess of the quantity which would be accounted for by the pus are found, nor is there any dropsy. There is increased frequency of micturition. Renal pain (nephralgia) and tenderness are nearly always present, but they vary widely in degree and character in the 3 varieties about to be mentioned. *The kidney should always be carefully examined* (§ 294), because, in addition to the renal congestion, all forms of pyelitis are liable to result in partial or complete obstruction of the infundibula and the gradual super-vention of hydro- or pyo-nephrosis. A few pus cells in the urine may be found in acute nephritis, after enteric and other fevers, and toxic doses of cantharides or turpentine. Apart from these there are 3 well-marked varieties or causes of acid pyuria.

1. **CALCULOUS PYELITIS** is due to the irritation set up by the presence of a stone. The *differential symptoms* are:—(i.) A history of renal colic (§ 302) is often present. (ii.) *Pain on one, the diseased, side*, which varies with exercise, and (iii.) hæmaturia, also varying with exercise. (iv.) The quantity of pus often

<sup>1</sup> Cases are recorded by H. E. Drake-Brockmann (Capt. I.M.S.), in *The Lancet*, 1900, vol. i., p. 1876.



varies from day to day; and the patient may feel easier after a discharge of pus, as the retained pus causes pain, and sometimes swelling. (v.) Attacks of intermittent pyrexia from time to time. (vi.) *Crystals in the urine* aid the diagnosis considerably.

“**Renal Tension**” may be attended by pus cells and blood in the urine; and in long-standing cases casts may also be present. In 1896, Mr. Reginald Harrison<sup>1</sup> drew attention to cases of patients who, presenting many or all of the symptoms of renal calculus, had been operated on without any stone being found, and who had nevertheless completely recovered from their symptoms after the operation. Such cases are by no means uncommon in the practice of most surgeons, and the kidney has generally been found to be enlarged and congested, and the capsule adherent. The anatomical condition is not uniform, and its pathological cause obscure. The commonest symptoms are *severe pain* in the loin, with hæmaturia, albuminuria, pyuria, and other urinary changes in varying proportion. Mr. Harrison recommends operation and reni-puncture in these cases and for cases of acute congestion or acute inflammation of the kidney in which death seems imminent from suppression of urine.

II. TUBERCULOUS PYELITIS. Tuberculous disease of the kidney always starts in the pelvis. It may be primary or secondary to tubercle elsewhere. Very often both kidneys are diseased. This condition may be very difficult to diagnose from Calculous Pyelitis, but the *differential symptoms* are:—(i.) no previous history of colic, but dull pain in the loins, liable to exacerbations from the passage of caseous masses; (ii.) hæmaturia is not usually present<sup>2</sup>; (iii.) the amount of pus in the urine does not vary, but steadily increases; (iv.) the urine contains amorphous granular matter and tubercle bacilli, but usually no crystals; (v.) *pyrexia of a regularly intermitting type*, with increasing emaciation; and (vi.) there are often evidences of tubercle in other parts of the body, as in the testes or lungs.

III. ASCENDING PYELITIS OR PYELO-NEPHRITIS arises from three groups of causes, which may conveniently be termed Obstruction, Extension, and Infection Pyelitis. (a) Some *obstruction in the urinary passages* below the kidney not infrequently causes retention and decomposition of the urine, and septic infection of the pelvis of one or both kidneys, which may go on to pyo-nephrosis (Fig. 91, p. 536). The diagnosis of this form, which used to be known as “Surgical Kidney,” rests mainly on the history of the

<sup>1</sup> Presidential Address at the Med. Society of London, 1896, and Presidential Address in the Surgical Section, Annual Meeting B. M. A., 1901.

<sup>2</sup> Occasionally hæmaturia is an early symptom of renal tuberculosis. Dr. Newman, *Lancet*, vol. II., 1899, p. 559.

cause of retention—enlarged prostate, urethral stricture, uterine and other tumours pressing upon, or calculus impacted within, the ureter (see also Retention, § 312). Here, as in the next group, the urine may be alkaline from concurrent cystitis. (b) Ascending pyelitis may also result from the *extension of cystitis* without obstruction, and thus the numerous causes of the latter disease (§ 305) are brought into operation, *e.g.*, gonorrhœa, septic catheterisation, etc.

(c) INFECTIVE PYELO-NEPHRITIS is a condition which is gradually being recognised by the profession. Previous bladder symptoms may be slight, transient, or altogether absent. The nature of the microbic infection which infects the urine is not always apparent; but that the *b. coli communis* is capable of so acting is beyond doubt. The *symptoms* of colus infection may be wholly indistinguishable from calculous pyelitis on the one hand and tuberculous pyelitis on the other, unless one is aided by the detection of the respective microbes in the urine. It seems possible that some of the cases referred to by Mr. Reginald Harrison (*supra*) were of this nature. There are, however, three features which in the author's experience are fairly characteristic of the colus infection: (i.) the occurrence of attacks of pyrexia at irregular intervals of a distinctly pyæmic type, attended by shivering, sweating, and vomiting; (ii.) a distinctive smell of volatile sulphides in the urine; and (iii.) the fact that pure cultures of *b. coli* can be readily obtained from a specimen of the urine collected through a sterilised catheter. The disease, which has so far only occurred in women, runs a most indefinite course, but usually wears itself out sooner or later. Anti-bacterial inoculations might be tried.

*Prognosis.* (i.) The most serious form of pyelitis is that due to extension of inflammation upwards from the bladder. When originating in gonorrhœal cystitis, death usually occurs in 7 to 14 days. (ii.) In the tubercular form there may be no general symptoms until the disease extends beyond the one kidney; in other cases it may be fatal in 12 to 18 months. (iii.) Calculous pyelitis may last indefinitely for years, though not without danger of uræmia and abscess of the kidney. (iv.) The course of ascending pyelitis depends very much upon the cause, the possibility of its removal, the age of the patient, and his general condition. In the last three forms *pyonephrosis* is apt to ensue.

*Treatment.* 1. In *all forms of pyelitis* fluid diet, milk and warm drinks, rest and warmth are essential; and cupping of the loins is sometimes useful. Sedative drugs, such as hyoseyamus and belladonna, may be administered, and antiseptics, such as boric acid, quinine and creasote may relieve the condition. Buchu, pareira, urotropin, and salol are useful. Many of these cases call for

nephrectomy or other surgical measures. The question is often raised (before or during operation) whether one or both kidneys are diseased. This is a difficult question to answer, and each case presents different data; but in all cases *daily observations on the amount of urea* should be made (cf. § 307). 2. Of *calculous pyelitis*. If due to uric acid calculi large doses of pot. cit. and bicarb. may be employed; if due to oxalates, nux vomica and nitro-hydrochloric acid; nephrolithotomy in nearly all cases. 3. Of *tubercular pyelitis*. Tonics such as iron, quinine and cod-liver oil must be given. Excision of the kidney is to be advised if (i.) the other kidney is believed to be healthy; and (ii.) there is no tubercular disease elsewhere in urinary tract, in the lungs or intestines. 4. For *ascending pyelitis*, our attention is best directed to the cause, and measures must be employed to antisepticise the urinary passages.

*A diminution in the specific gravity when marked and continuous, even in the absence of albumen, is suggestive of CHRONIC INTERSTITIAL, NEPHRITIS, or more rarely DIABETES INSIPIDUS. A marked INCREASE in the specific gravity is suggestive of DIABETES MELLITUS.*

§ 307. The other **causes of altered specific gravity** are relatively less important, because they are identified mainly by other means. Nevertheless, the sp. gr. of the urine is an extremely important feature, because, in the absence of sugar, it is a MEASURE OF THE UREA, the sp. gr. being higher in direct proportion to the amount of urea contained in a given sample of urine. Therefore, with certain reservations about to be mentioned, it is a very fair measure of the FUNCTIONAL ACTIVITY of the secreting substance of the two kidneys taken together. For example, when one kidney is known to be diseased or destroyed, it will give us a good idea of the condition of the other; and in chronic Bright's disease we may learn something of the amount of renal epithelium undamaged. In such cases regular estimations of the urea secreted should be made (§ 282). The reservations just alluded to are 4 in number—1stly, the sp. gr. must always be considered in relation to the total diurnal quantity of the urine; 2ndly, the total urea varies considerably with the body weight, being less in women and persons of slight build; 3rdly, it varies to some extent also with the amount of proteid food ingested,

and the work done by the body, being rather less in a person lying in bed; 4thly, it is assumed that the liver is healthy because, as mentioned in the introduction to Chapter XII., the first stage in the manufacture of urea takes place there; only the concluding stage being performed by the kidney.

The variations in the *total output of urea* have been mentioned under the several diseases of the kidney.

The specific gravity is DIMINISHED in—

1. Chronic Interstitial Nephritis.
2. Polyuria, and all the diseases about to be mentioned under that heading, excepting Diabetes Mellitus.
3. Myxœdema and other conditions where the nitrogenous disintegration within the body is diminished.

The specific gravity is INCREASED in—

1. Diabetes Mellitus (owing to the sugar).
2. Some renal diseases where the quantity of water is considerably diminished, such as Acute Nephritis, the Cardiac Kidney, etc.
3. Febrile and other conditions where the nitrogenous disintegration is excessive.
4. Whenever the urine becomes concentrated by profuse sweating, vomiting, or diarrhœa.

*An increase (POLYURIA), or diminution (ANURIA), in the quantity of urine is complained of by the patient in several important diseases.*

§ 308. In polyuria it is very desirable to measure the total diurnal quantity, since patients are very apt to mistake increased frequency for increased quantity, and *vice versa*.

There is INCREASED QUANTITY of urine secreted in—

1. *Diabetes mellitus*, which is known by the high specific gravity of the urine, and persistent glycosuria.
2. *Diabetes insipidus*—low sp. gr. and malaise, but no sugar.
3. *Chronic interstitial nephritis*, which is known by the low sp. gr. of the urine, slight albuminuria, etc. (§ 298).
4. *Waxy kidney*, which is known by the low sp. gr. of the urine, and great albuminuria (§ 299).
5. *Hydronephrosis*, which is known by the passage of large quantities of urine for a limited period of time, accompanied by the disappearance of a swelling from the loin. This is followed by a return to the normal both in quality and quantity of the urine, and then a gradual re-formation of the swelling (§ 316).
6. *Convalescence* after fevers.
7. *Temporary polyuria* occurs in hysteria, nervous excitement, and any other condition giving rise to reactionary or paralytic condition of the abdominal sympathetic.
8. During the administration of *diuretics*.



There is **DIMINISHED QUANTITY** of urine in—

1. Acute Nephritis.
2. Subacute and Chronic Tubal Nephritis (some stages).
3. Final stage of Chronic Interstitial Nephritis.
4. The Cardiac Kidney and some other Renal Congestions.
5. Febrile states.
6. Whenever there is profuse vomiting, diarrhoea or perspiration, or but little fluid is taken.

*The patient complains of polyuria; the urine is of HIGH SP. GR., and CONSTANTLY contains SUGAR (glycosuria); there are also thirst, and, in spite of a voracious appetite, gradual loss of flesh—the disease is DIABETES MELLITUS.*

§ 309. **Temporary glycosuria** may arise from a variety of transient causes, many of which are due to active hepatic hyperemia. Many of these causes are of little or no consequence. 1. Dietetic errors (glycosuria only after a meal). 2. Gradual occlusion of the portal vein (ditto). 3. In the reaction after large or prolonged doses of certain drugs: chloroform, chloral, morphia (the reaction here *may* be due to glycuronic acid). 4. After epileptic convulsions. 5. During the collapse of cholera. 6. During the paroxysms of ague. 7. In chronic Bright's disease with high tension. 8. Cardiac disease, asthma, pertussis, and some other cases of dyspnoea. 9. Injury to the liver. 10. Congestion of the liver in gouty people; and when much exercise is taken by those unaccustomed to it. 11. Intestinal irritation. 12. After concussion and compression of the brain, and tumour cerebri, especially if involving the floor of the fourth ventricle. 13. Violent mental and moral emotions. 14. During pregnancy and suckling. 15. Pancreatic disease. 16. After acute fevers such as cholera, malaria, influenza, or diphtheria.<sup>1</sup>

§ 310. **Diabetes Mellitus** is a constitutional disease, characterised by the passage of large quantities of urine containing glucose, associated with progressive emaciation, and voracious appetite. 1. There is usually increased frequency of micturition, and the patient passes large quantities (6 to 40 pints) of clear pale urine, which has a sweetish odour. If dropped upon the boot this leaves a crystalline deposit, by which means the condition has occasionally been identified. The specific gravity is high, 1030 to 1040 or more. The amount of sugar varies from 2 to 40 grains or more per ounce; and the total amount per day varies from 10 ozs. to 2 lbs. In diabetes the sugar may occasionally disappear for several days, but in general terms it is permanent and persistent. The diurnal quantity of urica and phosphates is increased; acetone may be present, and albuminuria sometimes, especially towards the end. 2. Progressive weakness and emaciation are sometimes the first symptoms to attract notice. 3. At other times thirst or voracious appetite accompanied by a raw beefy tongue and dry skin are the first signs. 4. The complications (*v. infra*) not infrequently lead to our detecting the disease, for its earlier stages are often overlooked by the patient.

<sup>1</sup> Cases of non-diabetic glycosuria are reported by Dr. Saundby, *B. M. J.*, April 14, 1900.

*Varieties.* There are two well-marked varieties of diabetes. a. The mild form which is met with in corpulent middle-aged people, where the symptoms are moderate, and dietetic restriction removes the sugar from the urine. This is really a *transient glycosuria*. b. The severe variety is met with in *acute* and *chronic* forms. The acute form usually occurs in children or young adults, and occasionally after head injuries. The chronic form is met with in older people, and is attributed sometimes to mental worry. It also occurs with tumour of the fourth ventricle, and other causes of transient glycosuria which become chronic.

*Causes.* Diabetes occurs in males, in the proportion of three to two, and may be hereditary. Gout, insanity or phthisis may be present in the family. Sedentary habits and brain overwork may predispose.<sup>1</sup>

The *complications* of diabetes are numerous. In order of frequency they are:—1. *Phthisis*, which is perhaps one of the most frequent causes of death in the condition. 2. Various *skin conditions*, especially eczema, boils, pruritus and xanthelasma, which appear early in the disease, and carbuncle and gangrene which appear in the later stages. It is an imperative rule to examine the urine for glucose in all cases of boils, carbuncles, and pruritus vulvæ. 3. The *nervous system* is specially apt to be affected, and peripheral neuritis is now known to be frequently caused by diabetes. The knee-jerks are commonly lost in diabetes, sometimes without any other nerve symptom, or there may be tingling, numbness, perforating ulcer, or neuralgia. Restlessness is common, and this may go on to mania or melancholia. The sudden supervention of diabetic coma often terminates life. 4. *Ocular changes* are almost as common as the foregoing, and these may take the form of soft cataract, or defective accommodation leading to a rapidly increasing presbyopia. Retinitis, optic atrophy, and amblyopia also occur.

*Diagnosis.* In any of the conditions mentioned under *complications* the urine should be examined; this is the key to the diagnosis. In *Diabetes insipidus*, *granular kidney*, *amyloid kidney*, and sometimes in *hysteria*, the quantity of urine is excessive, but

<sup>1</sup> Certain pathological and physiological considerations lead us to suspect that diabetes is due to disease of the liver or pancreas; but its pathognomonic symptom brings it clinically under urinary derangements, since its leading sign is a constant glycosuria.

in none of these conditions is sugar present. Two golden rules will often enable us to identify a case of diabetes which otherwise might be overlooked; 1, always examine the urine of a patient suffering from boils, or from eczema of the genitals, and 2, of a patient the subject of apparently causeless wasting. For the diagnosis of diabetes from *temporary glycosuria vide supra*.

*Prognosis.* 1. The mild form, which is met with chiefly in corpulent persons and others over 35, may occasionally pass into the graver form, but generally with suitable diet the sugar disappears, and the condition warrants an excellent prognosis. 2. In the severer forms the prognosis chiefly turns upon the age of the patient. If the disease is established in a young adult, life rarely lasts more than two years at the outside. The effect of *diet* is a valuable aid to prognosis. If by this means the sugar can be reduced to 3 or 4 grains per ounce the patient may live many years, especially if the disease did not come on until middle life was reached; if, on the other hand, 10 or 20 grains are constantly present, the case will go rapidly downhill. The presence of *complications* other than pneumonia or phthisis does not add very materially to the gravity of the situation. Death may ensue in three ways: (i.) by complications—a third of the cases die of phthisis; (ii.) by asthenia; and (iii.) about an equal number die with coma. Coma is heralded in most cases by certain symptoms which it is well to bear in mind—such as a decrease in the amount of urine, or the occurrence of albuminuria, or of diacetic acid in the urine (§ 289), epigastric pain, increasing languor, an accelerated and sighing respiration (“air-hunger”), and drowsiness. In some cases the coma supervenes suddenly, after a period of excitement. A peculiar sweet odour in the breath, when present, is a valuable means of diagnosis of diabetic coma.

The chief *treatment* is dietetic, and consists in the reduction of sugars and farinaceous foods (§ 214). Saccharin is taken in place of sugar. Among therapeutic agents codeine, one of the alkaloids of opium, still takes the first place. It is given in increasing quantities from gr.  $\frac{1}{2}$  to 6 or 8 grs. If diarrhoea is present opium may be given, and very large doses are tolerated. Uranium nitrate is a remedy still on its trial. Arsenic, bromides, and antipyrin are available, especially when the nerve structures are affected.

Among the symptomatic indications, thirst is best allayed by frequent doses of ac. phosph. dil.; the voracious appetite and dyspepsia may sometimes be allayed by bismuth. Coma has not hitherto been successfully treated by the ordinary means; but cases have been reported of at any rate partial recovery after large intravenous saline injections.

**Pancreatic Diabetes.** It is not quite certain whether this exists as a separate variety, but some cases of Diabetes Mellitus have been observed with pancreatic lesions, such as chronic inflammation, and fatty degeneration. They have been characterised by the presence of undigested fat in the faeces, and the ordinary symptoms of a severe diabetes running a very rapid course. In chronic pancreatitis and pancreatic calculi, glycosuria is often present. On the other hand, pancreatic disease may be unattended by glycosuria.

*The patient complains of polyuria and many of the other symptoms of Diabetes Mellitus, but the SP. GR. OF THE URINE IS LOW, and there is NO SUGAR—the disease is DIABETES INSIPIDUS*

§ 311. **Diabetes Insipidus** is characterised by great and persistent increase in the quantity of urine, without glycosuria or albuminuria, attended by great thirst and emaciation. It is believed to be due to a dilatation of the renal vessels, though how this permanent dilatation occurs is uncertain. The fact that the condition occurs with tumours of the medulla or pons, or with lesions involving the thoracic or abdominal nerve ganglia, is strongly suggestive of a vasomotor paralysis.

*Symptoms.* 1. The amount of urine may be very great, from 10 to 20 pints per day. It is pale in colour, so that it may resemble clear water. The specific gravity averages 1002–1005. The diurnal amount of solid constituents is as a rule not very much increased; and no other abnormality may be present. Occasionally traces of albumen and sugar appear towards the end. 2. In the mild form of the disease polyuria and thirst are the only symptoms; but in the severer variety nearly all the symptoms mentioned under diabetes mellitus are also present—dry skin, emaciation, large appetite, and alternating constipation and diarrhœa. Indeed, it is distinguished from that condition only by the absence of glycosuria. Intercurrent attacks of pyrexia have been observed. 3. Obscure nervous symptoms, with irritability of temper, are common in this disease—such as disturbed sleep, occipital headache, neuralgic pains in the lumbar region, diminished reflexes, and muscular twitchings.

*Diagnosis.* The disease is apt in its early stages to be mistaken for *chronic interstitial nephritis*, but the greater age of the patient, the presence of traces of albumen, and of cardio-vascular symptoms, and the absence of thirst and voracious appetite help to distinguish the latter condition. With *amyloid kidney* there is much albumen, and with both *hydronephrosis* and *cystic kidney* a tumour is generally palpable in the region of the kidney.

*Causes.* (i.) More males are affected (2 or 3 to 1 female). Childhood and early middle age are the favourite ages. (ii.) It is said to occur in association with a nervous temperament. Certainly among the chief



exciting causes are injuries to the nervous system, especially blows on the head. Intracranial tumour or inflammation or powerful emotions are also exciting causes. (iii.) Muscular exertion, exposure to cold, and intemperance are also mentioned.

*Prognosis.* The milder varieties may last for a great many years, and exist rather as an inconvenience than as a malady. In the severer forms, especially those due to intracranial tumours, the course may be very rapid, and death ensue in the course of a month. When setting in acutely after injury to the head (which may be attended by some glycosuria at first) recovery may ensue after a year or so. In children with the tubercular diathesis death usually occurs in the course of 1 or 2 years. In general terms, cases setting in acutely are more hopeful than those which start insidiously. Death may take place from gradual exhaustion, drowsiness passing into coma, with or without convulsions; or from complications such as phthisis or pneumonia.

*Treatment.* Most reliance is placed upon hygienic treatment. Tea, coffee, and other substances which increase diuresis should be avoided, but the amount of fluid taken should not be reduced below that excreted. Of drugs the favourite is valerian, gr. v. of the powdered root, increased up to gr. xxx. Nitroglycerine has been used; and arsenic relieves the gastro-intestinal and skin symptoms. When there is disease of the bulb, electricity may be tried—the positive pole placed on the back of the neck, the negative pole passed through the nostril to rest on the cervical spine,  $\frac{1}{2}$  to 5 milliampères for 5 minutes every second day.

*The patient complains that he cannot pass water, and a DISTENDED BLADDER can be made out by percussion and palpation above the pubes, or by the passage of a catheter—the condition is RETENTION OF URINE.*

§ 312. The causes of **retention of urine** come mainly within the province of the surgeon. Those of *sudden* onset are often due to urethral spasm or congestion; those of *gradual* onset are more numerous. The age and sex of the patient may aid us. Thus in *childhood* we may suspect impacted calculus, phimosis, or a ligature round the penis; in *women*, tumours pressing on the neck of the bladder—*e.g.*, fibroid or retroverted uterus, hysteria or reflex irritation after parturition; in young or middle-aged *adults*, stricture, gonorrhœa with congested mucous membrane, spasm after exposure to cold or a drinking bout, or tabes dorsalis; in *old men*, prostatic enlargement, or atony of the bladder. At all ages there may be calculus or tumour blocking the neck of the bladder, paralysis of the bladder from diseased or injured cord or brain, or reflex spasm after operations about the perinæum.

The *treatment* is mainly surgical, but in cases of spasm a hot

bath or hot fomentations to the abdomen will give relief. Hysterical and other nervous affections are referred to elsewhere. Atony and simple vesical paralysis may be treated by *nux vomica*, and the constant current, one pole being placed on the perinæum and the other just above the pubes.

*The patient complains that he has not passed any water for some time, but there are NO EVIDENCES of a DISTENDED BLADDER, and on passing a catheter it is found to be empty, or nearly so—the condition is SUPPRESSION OF URINE.*

§ 313. **Suppression of urine** is a very grave condition. A catheter should always be passed before the diagnosis of suppression is made. There are two kinds:—I. **OBSTRUCTIVE** suppression, which is due to some obstruction to the flow of urine through the ureters; and II. **NON-OBSTRUCTIVE** suppression, which is due to the non-secretion of urine by the kidneys. This latter form is sometimes spoken of as true suppression.

I. **OBSTRUCTIVE SUPPRESSION** is due to partial blocking of one or both ureters (the kidneys being healthy) by (i.) renal calculus; (ii.) tumour at the base of the bladder; (iii.) congenital malformation of the ureters. The *symptoms* are—(1) When only one ureter is completely blocked, the urine that passes is clear, of low specific gravity, and non-albuminous; and chronic uræmia ensues until the condition is relieved or the remaining kidney undergoes compensatory hypertrophy (see also hydronephrosis, § 316). (2) When both ureters are blocked a condition known as “*latent uræmia*” arises. The patient passes no urine for about a week, and may complain of nothing except slight drowsiness, but after 8 or 10 days he becomes restless, with contracted pupils, sub-normal temperature, dry brown tongue, and muscular twitchings. In other cases vomiting may be so severe as to suggest the presence of intestinal obstruction. Death is usually sudden, after 10 to 14 days, the mind remaining clear to the end.

II. The causes of **NON-OBSTRUCTIVE** or **TRUE SUPPRESSION** are: (i.) acute nephritis, or the terminal stage of chronic nephritis (10 to 20 hours before death); (ii.) collapse (of which suppression is one of the symptoms)—*e.g.*, after abdominal operations or injuries, passage of a catheter, fevers, or local inflammations; (iii.) hysterical

anuria; (iv.) acute lead, phosphorus, or turpentine poisoning; (v.) embolism or thrombosis of both renal arteries (very rare). Whichever of these causes is in operation, the *symptoms* are: (1) any urine passed is high-coloured and concentrated (high specific gravity), and may contain albumen and casts (indicating that the suppression is due to renal disease); (2) there may be urgent vomiting, diarrhœa, and sweating. The other symptoms vary according to the cause, but in general terms are those of acute uræmia (§ 273).

*Prognosis.* Suppression is a very serious symptom, though the gravity depends somewhat upon the cause. Of the *obstructive* forms, calculus blocking one ureter, the kidney of the opposite side being healthy, is perhaps the most favourable. If the obstruction affects both ureters and is not removed, death will occur in about eleven days after the obstruction began. In the *non-obstructive* forms death or partial recovery takes place in a few days.

*Treatment.* Hot baths, pilocarpine, and other diaphoretics promote the action of the skin and so relieve the toxæmia. Free purgation promotes the excretion by another channel; cupping, wet or dry, over the loins relieves the local congestion. For the treatment of obstructive suppression a surgeon should be called at once. Stone is usually impacted in the ureter at its entrance to the bladder.

*The patient complains that his urine dribbles away constantly, and on percussing over the pubes or passing a catheter his bladder is found to be empty—he has TRUE INCONTINENCE. Or he complains that he has a frequent call to urination and cannot always hold his water—he has ACTIVE INCONTINENCE.*

§ 314. **Incontinence of urine** may be of two kinds, and it is best to speak of these as TRUE INCONTINENCE and INCREASED FREQUENCY (active incontinence) respectively.

(a) TRUE INCONTINENCE, when the urine dribbles away involuntarily as fast as it is formed, must not be confused with *overflow* or *false incontinence*, which is due to the overflow of a distended bladder in *retention*. The latter is recognised by the percussion signs of a full bladder, and by the relief afforded by the passage

of a catheter. In true incontinence, which is relatively a rarer condition, the *cause* is generally quite apparent, such as vesico-vaginal fistula, paralysis and dilatation of the sphincter after the operation of lithotrity, or the paralysis of the sphincter associated with various cerebro-spinal affections.

(b) INCREASED FREQUENCY OF MICTURITION, or as it is (unfortunately) sometimes called, *active incontinence*, is a very common complaint. The patient can hold his water, but the calls to urinate are too frequent, and sometimes so urgent that a few drops dribble away before arrangements can be made. The normal time during which the urine can be retained differs in different individuals, and also according to the amount of fluid taken ; but four to five hours is a fair average. It is longer in the female than the male ; some women can retain the urine for ten or twelve hours. The habit is injurious, and is said to lead to flexions of the uterus.

Increased frequency is due to a variety of *causes*. The first thing to determine is whether there is any marked increase in the diurnal quantity, as in diabetes or chronic granular kidney, because any of the causes of polyuria (§ 308) may be a cause of increased frequency of micturition. In young adults diabetes is perhaps the commonest, but in advancing years granular kidney and enlarged prostate are by far the most common causes. Indeed, our attention is often first drawn to the latter condition by the fact that the patient develops a habit of rising at night to pass water. It is not always easy to decide whether the quantity is increased or not, as the patient is apt to think that, because he passes water too often, he passes too much. But having as far as possible excluded polyuria, there remain three groups of causes to consider. 1. Some cause of *local irritation* is undoubtedly the most frequent. The *urine* may be too acid. The *bladder* may be irritable, as from the presence of an enlarged prostate (the usual cause of abnormal frequency in old age), chronic cystitis, ulceration, tumour, stone (in the young), or pressure upon the viscus by a displaced uterus. Or the irritation may be in the *kidneys* from the presence of stone, tubercle, or other cause of pyelitis (§ 306). Or the irritation may be *reflex*, from disease in the vicinity of the bladder, worms, phimosis, or too long a prepuce (a very



frequent cause of nocturnal incontinence in children), fissures, piles, prolapse or polypus of the rectum, vascular urethral caruncle (a cause frequently overlooked in women), pelvic inflammation, or varicocele. 2. *Constitutional* causes are occasionally associated with this condition, such as hysteria, sexual excesses, nervous debility, adenoid vegetations in the pharynx, and other causes leading to deficient aeration of the blood. 3. A *congenital* want of development of the sphincter is sometimes present. True congenital cases are rare, and defective action of the sphincter is more frequently due, especially in women and children, to some of the reflex causes above mentioned, the habit persisting after the cause has been removed.

NOCTURNAL INCONTINENCE in children is a troublesome condition often met with in private practice. In such cases we must first satisfy ourselves of the absence of any organic disease. Having done this it is well to remember in this condition that it may be associated with incipient insanity in childhood, general debility, stone in the bladder, and adenoid vegetations in the pharynx. The last-named, if severe, result in a deficient aeration of the blood and an unduly heavy sleep, with consequent insufficiency of the sphincter to control a full bladder. The other causes mentioned above should also be remembered.

Both *prognosis* and *treatment* turn almost entirely upon the cause, and are hopeful in proportion as this is removable. The power of retention of the urine is a habit which can be cultivated in early life, and the relative frequency in different individuals varies a good deal with habits engendered in childhood. Careful local examination should always be made to exclude local causes. If the urine is acid, or the bladder irritable, much good may be done by the administration of alkalies and hyoseyanus. If the bladder is wanting in tone, belladonna and nux vomica are the two sovereign remedies. Tinct. rhus aromat. ℥ v. to xv.<sup>1</sup> has been found to be useful where no cause is obvious. If there is irritability of the nervous system bromides are specially useful.

Children of faulty habits may be treated by sleeping on hard mattresses, or by preventing them sleeping on the back by means of a reel of cotton fixed to the sacrum by plaster. The amount

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<sup>1</sup> Dr. Freyberger, *vide* "Treatment," May 12, 1898.

of fluid should be diminished, and they should be made to pass water before going to bed. Raising the foot of the bed, and cold douching to the spine are recommended.

§ 315. *The urine presents a cloudiness, due to some CRYSTALLINE or OTHER DEPOSIT*—it may be URATES, URIC ACID, PHOSPHATES, OXALATES, or FAT; unless it be pus (§ 290), blood (§ 285), or bacteria (§ 290).

In **lithuria** the urine, CLEAR when first passed, becomes cloudy with a pinkish AMORPHOUS DEPOSIT when it gets cold; the deposit dissolving again when heated in a tube. The condition described as LITHÆMIA (the clinical condition associated with lithuria) is still by most believed to be due to functional derangement of the liver, and its symptoms are described in the disorders of that organ (§ 251). Various other conditions with which excess of urates and uric acid in the urine may be associated, as a more or less subordinate symptom, have already been referred to in § 293.

The clinical significance of uric acid and urates is still a subject of debate.

**Phosphaturia** is usually indicated by cloudiness in a neutral or alkaline urine (§§ 287 and 293). 1. Phosphates frequently occur in the urine in such quantity as to cause a turbidity even when first passed. They are apt to occur especially towards the end of micturition, not infrequently alarming the patient unnecessarily. Phosphates may be especially abundant in the "alkaline tide" of the early morning, or after dinner. There may be no symptoms, even when phosphates are passed in large quantities: but more frequently phosphaturia is accompanied by chronic dyspepsia, or some condition in which the urine is alkaline. Phosphates are thought by some to be an evidence of excessive nerve waste: I have seen several cases of phosphaturia in medical men who had recently undergone severe brain work and nerve strain.

2. The name PHOSPHATIC DIABETES has been given to a condition where there are thirst, emaciation, aching pain in the loins and back, and an increase of phosphates in the urine, the diurnal quantity of which is greatly increased. The urine is alkaline or very feebly acid in reaction. In diabetes mellitus the phosphates vary inversely as the sugar. Phthisis may supervene, or the disease may pass into diabetes mellitus or diabetes insipidus, if unrelieved by treatment.

3. There is an increase of phosphates in wasting diseases, in leukæmia, severe anæmias, and in convalescence from fevers.

4. Phosphates are diminished in acute fevers, and in diseases of the kidney, e.g., nephritis.

5. *Stellar phosphates* may indicate grave constitutional disturbance: e.g., diabetes and cancer. *Triple phosphates* found in freshly passed urine denote that decomposition is going on in the bladder, an indication of cystitis. It is liable to deposit within the bladder or to form stone.

The *treatment* of "phosphatic diabetes" is by rest, warmth, and light nourishing food. Alcohol and coffee should be forbidden, as they promote diuresis. Codeia or opium should be administered until pain is abated; when tonics, iron, quinine, nux vomica, and cod-liver oil should be freely given.

**Oxaluria** is generally indicated by a "powdered wig" deposit on the top of the mucus which settles to the bottom (§ 293). Transient oxaluria has no clinical significance except as indicating the nature of a stone, which has revealed its presence by other symptoms. It is also found after a diet of rhubarb, tomatoes, cabbage, or onions. But oxaluria is also connected with four other clinical conditions.

(i.) The *oxaluric diathesis* is a tendency which exists in some individuals to the excessive formation of oxalates in the urine. Certain cases have been recorded where the symptoms of rapid emaciation and pains in the loins and back were attended by an excess of oxalates in the urine.<sup>1</sup> (ii.) Other observers have connected certain nervous symptoms, such as mental depression going on to neurasthenia and even melancholia, with this condition.<sup>2</sup> It is probable, however, that these symptoms are connected with the concurrent dyspepsia. (iii.) Oxaluria very often seems to be connected with dyspepsia. Urates are generally precipitated in the urine at the same time as the oxalates, and Sir Lauder Brunton has shown that the passage of sulphuretted hydrogen through a strong solution of urates gives rise to the formation of oxalates, by its reducing or deoxidising power. In intestinal dyspepsia a large quantity of this gas is formed in the intestines, and it seems probable that oxaluria in these cases may be caused by the deoxidising or reducing power of the  $H_2S$  upon the urates. (iv.) Oxalates are found in large excess in paroxysmal hæmoglobinuria (§ 303).

**Fat** may occur in the urine in chronic tubal nephritis attended by much fatty degeneration of the epithelium, and after fractures of the bones. It is found in great abundance in **Chyluria**. The presence of chyle in the urine gives to it a milky white appearance and the power of coagulating. Chyluria is not uncommon in the tropics where it is due to the migration of the *filaria sanguinis hominis* from the lacteals into the urinary tract, the unnatural communications thus made leading to the paroxysmal appearance of chyle in the urine. The urine passed at night is the more completely white; that passed by day may be mixed with blood. The embryos of this parasite are to be found in the urine with a few red and white blood cells, albumen, fat, and shreds of fibrin. However, other cases have been observed in persons who have never resided in the tropics, and the causation of such cases is obscure.

*Prognosis.* The patient may live 20 years with but little impairment of health. In other cases, however, great debility and mental depression may be present.

*Treatment.* Prevent the disease by boiling the drinking-water. Gallic acid is recommended. To meet the drain on the system give plenty of food.

§ 316. **Renal Tumours** may be of six kinds:—(I.) **HYDRO-NEPHROSIS**; (II.) **PYONEPHROSIS**; (III.) **PERINEPHRIC ABSCESS**; (IV.) **MALIGNANT DISEASE**; (V.) **CYSTIC DISEASE**; and (VI.) **MOVABLE KIDNEY**. The last-named comes under **Abdominal Pain** (§ 178), which is its chief symptom.

The *physical signs* common to all tumours of the kidney, and their diagnosis from other **ABDOMINAL TUMOURS**, are given in §§ 189 and 294.

<sup>1</sup> Cantoni, "Oxaluria." German translation by Hahn; Berlin, 1880; Begbie Schmidt's *Jahrbh.* lxxvii. 62, 1880; and Jaksch, *op. cit.*, p. 307.

<sup>2</sup> Neldert, *Munchener Med. Wochenschr.* xxxvii. 590, 1890.

**I. Hydronephrosis** is a term indicating a cystic tumour of the kidney, caused by the gradual obstruction of the urinary passages, and the consequent dilatation of the pelvis of the kidney.

The *symptoms* by which this tumour is recognised, are :— (1) at intervals a large amount of urine passes, with concomitant reduction or even disappearance of the tumour. The urine is pale, clear, and of normal composition. (2) Constitutional and general symptoms may be absent. (3) Local pressure symptoms may arise, causing pain or disturbance of function of the neighbouring organs.

*Etiology.* The causes of obstruction to the outflow of the urine may be :—(i.) *congenital* (contracted or twisted ureters); (ii.) *acquired* causes, which may occur (a) in the *urethra*, such as stricture or enlarged prostate (Fig. 91); (b) in the *ureter*, such as occur from stone or blood clot; pressure by pelvic or other tumours; contraction after operation injury or disease of the ureter; or kinking, as in movable kidney. These acquired causes give rise to a gradual obstruction, and when the obstruction is intermittent the tumour

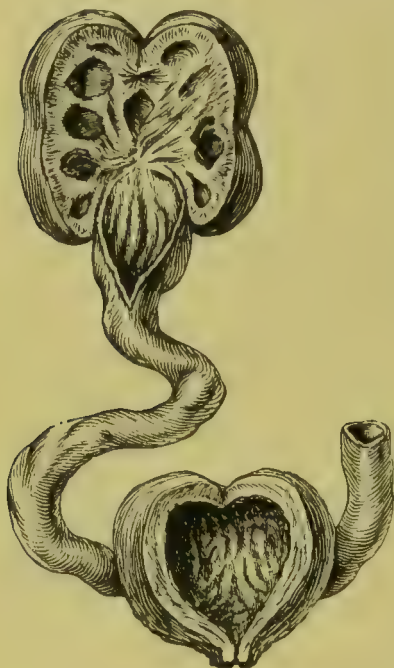


Fig. 91.—Results of URINARY OBSTRUCTION such as arise from enlarged prostate or stricture urethræ (Walsham's Surgery).

may become very large. It is then liable to be mistaken for an ovarian cyst, or even for ascites. In such cases a trochar may be introduced, and the fluid withdrawn would reveal an absence of the albumen which is always present in an ascitic fluid.

*Prognosis.* If the condition is unilateral and intermittent it may cause little trouble, and may disappear after a duration of years. On the other hand, a double hydronephrosis is very serious, as it leads to uræmia. The complications are :—rupture into the peritoneum or pleura; the onset of suppuration in the pelvis of the



kidney (pyonephrosis); or uræmia, due to atrophy of the substance of both kidneys.

*Treatment.* If the tumour is intermittent, unilateral, and causing few symptoms, it is best to leave it alone. Osler recommends the use of a pad to retain the organ in place and prevent further dilatation.<sup>1</sup> If the tumour becomes very large, surgical treatment is advisable. In all cases the cause must be ascertained and, if possible, treated.

**II. Pyonephrosis** is a cystic tumour of the kidney due to distension of the pelvis and calices, by fluid containing pus or purulent urine. It is consequent on obstruction to the free outlet of the urine in septic cases of pyelitis, or sepsis supervening on hydronephrosis.

The *symptoms* are:—(1) the tumour is tender to palpation; (2) symptoms of pyelitis are present—pyuria, intermittent pyrexia, sometimes rigors, and dull pain in the loin; (3) at intervals, when the obstruction is removed or diminished, the tumour may subside, coincident with the passage of a large quantity of pus in the urine.

The *causes* are:—(1) *pyelitis* (§ 306) with blocking, partial or complete, of the ureter; or (2) *hydronephrosis* (*vide* causes of this above) becoming septic, *e.g.*, from extension upwards of cystitis.

*Diagnosis.* (1) From *hydronephrosis*, which has no tenderness or fever; (2) from *perinephric abscess*, which has greater tenderness in the loin and a more superficial swelling, with local signs of abscess sooner or later.

*Prognosis.* The condition is very grave. A tubercular pyonephrosis may undergo cure by caseation; but in most cases the patient becomes worn out with the long discharge, or develops amyloid disease, or a fatal issue is rapidly brought about by the tumour bursting into the abdomen or chest.

*Treatment* is mainly surgical, and nephrotomy is indicated. The cause must be treated medically.

**III. Perinephric Abscess** is not very uncommon. It may arise by (i.) extension from kidney disease (pyelitis); (ii.) extension from a perityphilitic abscess; (iii.) extension from other organs—*e.g.*, abscess of the liver or Pott's disease of the spine; (iv.) after an injury. The *symptoms* are: (1) dull, aching pain in the loin radiating down the leg; (2) deep-seated resistance in the hypochondrium in front, tender to pressure; (3) the

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<sup>1</sup> Professor William Osler, "Abdominal Tumours," 1894.

temperature is continuous or pyæmic in acute cases with sudden onset, or intermittent in insidious cases: (4) the leg on the same side is kept flexed; (5) swelling, which appears late in the disorder, it is seen between the iliac crest and the last rib, and it may be fluctuant; (6) the urine may or may not be altered according to the cause, but traces of albumen are common. The *diagnosis* is difficult in the early stage when pain alone is present, when it may readily be mistaken for *lumbago* or *spinal disease*, but there is no fever in the former. Later it may be mistaken for a *renal tumour*, but in a simple tumour fever is absent, and the leg would not be held constantly flexed. In *pyonephrosis*, there is not such acute pain or tenderness. *Prognosis*. The abscess tends to open or to burrow its way in various directions, into the alimentary or urinary canals, peritoneum, or pleura. *Treatment*. In the early stages, before the diagnosis can be certain, give hot fomentations and opium for the pain; as soon as pus is recognised operative procedure is necessary. It may point in the lumbar region or various other directions, and burrow for a considerable distance.

**IV. Malignant disease starting in the kidney** is certainly a rare condition, as it has only been found in about 1 in 500 autopsies on persons dying of malignant disease.<sup>1</sup> It affects children under 9 (in whom *sarcoma* chiefly occurs), and adults over 40 (in whom it is mostly *carcinoma*), there being a remarkable immunity between these age periods.<sup>2</sup>

The *symptoms* are:—(1) The tumour is rapidly growing, usually of firm consistence, but if of very rapid growth it may appear fluctuating; (2) hæmaturia, frequent, intermittent, and of moderate amount; (3) progressive emaciation; (4) the pain is variable, sometimes it is very severe, owing to pressure upon or infiltration of the neighbouring organs. Sometimes pain is entirely absent, and the tumour may have attained a very large size before any symptoms occur.

*Causes*. Renal sarcoma is the commonest abdominal growth in children, and it is believed often to start before birth. According to Bland Sutton, it is met with in the first five years of life, and then, after a period of immunity, is found again in people between 50 and 60.<sup>3</sup> In people over 40 cancer is the most common form of malignant tumour.

*Diagnosis*. When a tumour occurs in a movable kidney it is apt to be mistaken for *ovarian tumour* or *fibroid*, and vaginal examination is necessary to clear up the diagnosis. *Tubercular* kidney in a child may present difficulty, but it has less pain, and pyuria is present rather than hæmaturia. *Pyonephrosis* is accompanied by fever, the swelling is fluctuant, and there is a history of pyuria. The diagnosis of malignant tumours is not usually difficult.

The *Prognosis* is very grave. If untreated death occurs in 6 to 12 months after detection of the growth, the cancer of adults being somewhat more slowly growing.

*Treatment* is usually too late; early excision gives the only chance of life. Medical treatment is entirely palliative.

<sup>1</sup> Discussion on Renal Tumours, Path. Sec. B. M. A., 1899.

<sup>2</sup> The solid tumours affecting the kidney consist of (A) *Connective tissue type*:—I. Simple or benign growths (fibroma, lipoma, angioma); II. Malignant Sarcoma, which is by far the commonest. (B) *Growths of an epithelial type*:—I. Adenomatous growths (simple adenoma, trabecular and papilliform cystomata); II. True Carcinoma:—(1) glandular type; (2) malignant papilloma. (C) Adrenal inclusions. (D) Adrenal growths.—“Renal Growths,” T. N. Kelyack, Manchester.

<sup>3</sup> *Clin. Journ.*, May 24, 1899.

**V. Cystic Disease of the Kidneys**<sup>1</sup> is a rare condition usually of congenital origin in which both kidneys contain cysts of varying size and number.

*Symptoms.* (1) There is a swelling usually in both lumbar regions, of insidious growth, very hard at first, and later yielding. (2) The other symptoms are similar to those of chronic interstitial nephritis—the urine is abundant, pale, of low sp. gr., containing traces of albumen, and occasionally blood and casts. The heart becomes hypertrophied, and the pulse indicates high tension. The patient may have excellent health for many years, or may develop symptoms of chronic uræmia.

The *diagnosis* may be difficult. When symptoms of granular kidney occur, together with a tumour in both renal regions, the condition may be diagnosed as Cystic Kidney. The tumours have to be diagnosed from other abdominal tumours (§ 189).

*Causes.* The disease may occur in the fetus. Patients with the above symptoms, however, are usually men, over middle age. Out of 21 cases collected by Dr. W. H. Dickinson, 11 were over forty.

*Treatment* is similar to that of Bright's disease. Death may occur from uræmia or the same complications as those of chronic interstitial nephritis.

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<sup>1</sup> *Varieties*—(i.) The cystic kidney in its typical form is a mass of cysts, and is usually congenital; (ii.) cystic kidney may arise in connection with granular kidney; in this variety the tumour is never so large as in the former; (iii.) cystic formations may also be due to hydatid.

## CHAPTER XIV.

### DISEASES PECULIAR TO WOMEN.

THE symptoms and consequences which may arise from disorders of the female genito-urinary organs are very numerous and widespread. Indeed, there is hardly a physiological system which does not suffer when these organs become affected. It is perhaps on this account that they should receive more attention from the general physician than is the custom.

#### PART A. SYMPTOMATOLOGY.

§ 317. The symptoms proper to these organs may be divided into *local* and *general*. The LOCAL SYMPTOMS are—certain external conditions around the vaginal orifice, leucorrhœa (vaginal discharge), dysmenorrhœa (painful menstruation), menorrhagia (excessive menstruation), amenorrhœa (deficient menstruation), pain in and around the organs, various disorders of function (*e.g.*, dyspareunia and dysuria), and tumours of the uterus.

The GENERAL SYMPTOMS consist of (1) malaise and general ill-health, which is often quite out of proportion to the amount of local mischief. A life of chronic invalidism not infrequently supervenes upon some chronic though slight derangement of the reproductive organs. This general weakness is specially apt to affect the nervous system, and one is sometimes tempted to credit the older authors who named hysteria on account of its supposed origin in the womb (*ὑστέρος*). (2) "Dyspeptic" symptoms of a reflex kind are nearly always present, as in other disorders connected with the abdominal viscera. (3) Anæmia is another consequence, though this may be due in part to the confinement indoors, or to the "loss" in cases of excessive menstrual flow. (4) Various neuralgiæ, and a general hypersensitiveness of the sensory and sensitive apparatus. A certain degree of this is normal during the menstrual periods, and as civilisation advances



it seems as though this recurrent hypersensitiveness were increasing. But by degrees, especially in those who suffer from dysmenorrhœa, this undue generalised hyperæsthesia is prolonged into the intervals between the periods.

**Case-taking** in diseases of women differs somewhat from that given in Chapter I. The following summary will form a guide to the principal questions to be answered as a matter of routine :—

(1) What is the leading symptom complained of by the patient ?

(2) History—name, age, married or single. (a) If married, how long ? How many children ? Date of last confinement ? Any miscarriages ? Confinements easy or difficult ? How long in bed after the birth ?

(b) Menstruation—age at which it commenced ? (i.) Regular ? 28 day or 30 day type ? Lasting 3, 5, 7 days ? (ii.) Blood coming in clots (means excess) ? (iii.) Painful or not ? Pain dated back to a particular time ? Pain in small of back, shooting down one or both legs, or in ovarian region ? Pain persistent or paroxysmal ? What relation to the flow ?

(c) Any intermenstrual discharge—Duration ; quantity ; white, clear, or thick and yellow ; offensive ; or with *débris* and blood.

(d) Micturition—Painful, dribbling or too frequent. Condition of bowels—pain on defecation ?

(e) Other physiological systems to be inquired into ; and whether general health has suffered.

#### PART B. PHYSICAL EXAMINATION.

§ 318. With the exception of certain circumstances, an abdominal and local examination should be a matter of routine in all gynæcological cases which are not on the surface obvious.<sup>1</sup> There are four methods by which the female pelvic organs can be investigated.

(a) AN EXTERNAL EXAMINATION of the abdomen—inspection, palpation, percussion, auscultation (Chapter IX., § 167).

(b) A VULVO-VAGINAL EXAMINATION should not be undertaken without duly considering both the necessities of the case, and the feelings of our patient (p. 543). A right-handed couch is indispensable so that the physician may be on his patient's right. The patient should lie on her l. side, with left arm out behind her, and the legs both drawn up ; the light should come from the foot of the couch. Note by inspection the colour and condition of the vulva,

<sup>1</sup> Some years ago I was urgently called to see a young lady who had been taken very suddenly ill with severe pain in the abdomen, whom I found rolling about the floor. Her history was that for some 9 months she had complained of menstrual derangement. She had been under the care of many different practitioners of both sexes, but had persistently refused to allow a thorough local examination to be made. In the presence of such circumstances as these I deemed it unnecessary to argue the point with my patient, lifted her on to the bed, and after making a thorough and complete examination in every direction diagnosed rupture of a large ovarian cyst. This was removed by a surgeon in the course of the next few hours, and thus the patient's life was saved. I mention this case because, although it is sometimes unnecessary to make a local examination, it is always desirable to investigate matters thoroughly.

hymen, urethral orifice, and the condition of the perineum, especially in women who have borne children, and then proceed to pass the finger gently. Some use the first, others the second finger; it is useful to be able to use either hand so that we may keep one hand for possibly septic cases alone. The finger nails should be kept extremely short and smooth, both for comfort to our patient and cleanliness.<sup>1</sup> As a lubricant for the finger some physicians like sanitas with vaseline (about 5 per cent.), others use carbolised glycerine (1 in 200). The finger is passed well in, and the condition of the vaginal walls noted; the position and condition of the os, whether patulous and soft as in pregnancy, firm, granular, fissured, conical, etc. Note also any fixity of the uterus, and whether there is an angle or dip between the cervix and body anteriorly or posteriorly such as occurs in flexions.

(c) It is very desirable to make a BIMANUAL EXAMINATION next in order. Instruct the patient to turn over on to her back, draw her right leg up, and, with the finger of the right hand still in the vagina, place the left hand firmly above the brim of the pelvis so as to be able to manipulate the uterus between the two hands. In this way note the size, position, and mobility of the uterus, the presence or absence of tumours, displacements of the uterus, or pelvic swellings or exudations.

(d) Various INSTRUMENTS are of considerable aid.

(1) The *sound*—invented by the same brilliant genius that adapted chloroform for use in surgical operations (the late Professor Simpson, of Edinburgh)—is the most valuable of all instruments. Its use, however, is contra-indicated in:—(i.) pregnancy, (ii.) menstruation, (iii.) acute inflammation in the pelvis, (iv.) cancer; and (v.) it should never be passed before making a bimanual examination.

With the tip of the right forefinger against the os pass the sound along the palm of the right hand until it slides well into the cervix. Then by a gentle turn and by a very gentle pressure upwards the sound will pass upwards and forwards into the uterine cavity.

The uses of the sound are to discover:—(1) the depth of the uterus, which is normally  $2\frac{1}{2}$  in., and the thickness of its wall; (2) the direction of the uterine cavity, whether retro- or ante-flexed, or pushed to one side by pelvic growths; (3) the state of the endometrium; (4) the size of the os; (5) the presence of tumours in the uterus.

<sup>1</sup> A story is told of the late Mr. Lawson Tait, whom a great many foreigners used to visit. One particularly insistent gentleman, who generally had long dirty nails, was always seeking to ascertain from him the secret of his success. Lawson Tait, who was not in the habit of measuring words, became somewhat annoyed at the insistence of his visitor, and one day in reply to the oft-repeated question he said, "The secret of my success is that I keep my nails short and extremely clean."

(2) *Vaginal speculum*. Before passing a vaginal speculum in single women the late Dr. Matthews Duncan advised that the consent of three persons should be obtained—(1) that of the patient; (2) that of her parent or guardian; and (3) that of the physician.

Many different specula are in use, as most gynecologists of eminence invent a new one. Practically they are of three types. The Ferguson, which is a tube; the bivalve or trivalve, which consists of 2 or 3 limbs jointed together; and the duckbill, which consists of 2 separate pieces. The first is best for the examination of the os; the second for the examination of the walls of the vagina; and the third for operative measures. In passing it do not forget the vaginal canal is directed backwards and upwards, and less pain is produced by quick movements in the right direction than by slow bungling. Note the condition of the mucous membrane, and the character of any discharge. If it be desirable to make some application to the interior by means of a Playfair's probe, this should be done before withdrawing the speculum.

(3) The  *volsellum*  is a hook for drawing down one or other lip of the os, which is desirable, for example, (i.) for the introduction of tents. It is also of use to examine (ii.) any catarrhal patch, (iii.) whether the uterus is freely movable, and (iv.) to palpate the posterior surface of the uterus. It is contra-indicated in those conditions in which the sound is contra-indicated, and also in tubal pregnancy.

DILATATION OF THE CERVIX may be done by 2 methods :—

(1) *Slow Method*. Sea tangle, tupelo, or sponge tents are inserted into the os uteri, and left *in situ* for some hours. By the absorption of fluid they swell up and distend the cervical canal. This method is useful in nulliparous women, or when the cervix is rigid.

(2) *Rapid Method*. Hegar's dilators are employed. These are heavy vulcanite or metal instruments of various sizes, which are useful in multipara when the cervix is soft, *e.g.*, after a confinement or abortion. It is desirable to do it under chloroform. Having inserted the duckbill speculum, fix the anterior lip of the cervix with the volsellum drawn well down, or by ovum forceps, and insert the dilators gradually one after the other until the cervix is large enough to examine the interior with the finger. In this way one can (1) examine the state of the endometrium by the finger; (2) curette the interior if there is any granular endometritis, or (3) diagnose the nature of any growth present by a microscopic examination of the scraping (a procedure which should never be omitted), thus differentiating, for instance, between cancer and simple endometritis. Dilatation of the cervix is contra-indicated in tubal disease, possible pregnancy, or cancer of the cervix.

#### PART C. DISEASES OF WOMEN, THEIR DIAGNOSIS, PROGNOSIS AND TREATMENT.

§ 319. **Routine procedure and classification.** Having ascertained the patient's principal or *leading symptom*, and the leading facts as to the *history*, according to the scheme given in Part B., proceed, unless the nature of the case is not already apparent, to the *physical examination* (subject to the reservations mentioned in Part B.).

CLASSIFICATION. The diseases of the female reproductive organs may be arranged, like urinary disorders, under the various cardinal symptoms to which they give rise, viz. :—

(a) Diseases of the Vulva and external parts . . . . .	§ 320
(b) Leucorrhœa . . . . .	§ 321
(c) Dysmenorrhœa . . . . .	§ 322
(d) Menorrhagia . . . . .	§ 323
(e) Amenorrhœa . . . . .	§ 329
(f) Pelvic pain, acute § 331, chronic . . . . .	§ 336
(g) Pelvic Tumours . . . . .	§ 337
(h) Painful sitting, Dispareunia, Dysuria, and other disorders of function . . . . .	§ 341

§ 320. Diseases of the vulva are generally surgical and can only be enumerated here.

(1) VULVITIS in children may be caused by the migration of round worms, by uncleanness, debility, or bad habits. In adults it is generally accompanied by vaginitis (*q.v.*).

(2) PRURITUS VULVÆ (itching) is sometimes a very troublesome condition. An examination should always be made to discover whether eczema, pediculi, or irritating discharges be present. If these be absent diabetes may be suspected. It also occurs in old age and in neurotic cases.

(3) CARUNCLE is a minute red irritable papilloma situated most usually just within the urethral orifice. It is a frequent cause of painful micturition, painful sitting, and painful coitus. Careful examination is needed for its discovery.

(4) LABIAL THROMBOSIS is readily recognised and is a not infrequent condition in certain hyperinotic states.

(5) ABSCESS of the vulva sometimes follows the last named. Sometimes it occurs as an inflammation of Bartholini's gland.

(6) NOMA, DIPHTHERIA, CHANCRES, CONDYLOMATA, ULCERS (simple or malignant) also affect the part.

In the *treatment* of vulval conditions cleanliness is essential, and on the whole the lack of this is one of the most frequent causes of vulvitis. It is surprising what little attention is paid to this matter, as is shown by the immense quantities of epithelial cells which are habitually found in the urine. Any eczematous or local condition must be treated as elsewhere. Caruncle is best treated by strong nitric acid or Paquelin's cautery. Labial thrombosis requires rest. Pruritus vulvæ may in my experience often be



cured by large doses of calcium chloride. Cases which have long resisted other treatment have yielded to this.<sup>1</sup> Locally, lotio calaminæ co. in weak carbolic acid solution, liq. carbon. deterg., and soda bicarb. and borax solution are employed in varying conditions. In cases where the itching is very intense, a solution of nitrate of silver (20 grs. to the ounce) may be painted on, the parts having been first anæsthetised by the application of cocaine solution. At the same time internal remedies, such as arsenic, quinine, bitter tonics and calcium chloride should be gone on with.

§ 321. **Leucorrhœa** is any white or whitish discharge from the vulval orifice. It is colloquially known as the "whites." There are three pathological causes of leucorrhœa—vaginitis, endocervicitis, and endometritis. When it comes from the *vagina* it is a thick, opaque white fluid, containing flocculi. If it comes from the *cervix* it has a more glairy and tenacious character, but is fairly constant, resembling in that respect the vaginal discharge. But if it come from the body of the *uterus* it is "curdy" and comes in gushes when the patient sits up or moves about. Examination by the speculum settles its origin, and is generally necessary for correct diagnosis.

(a) LEUCORRHŒA OF VAGINAL ORIGIN arises when there is vaginitis from any cause, either acute or chronic.

(a) In ACUTE VAGINITIS the discharge is profuse, yellow or greenish, and sometimes blood-stained, attended by dysuria and local signs of inflammation. The chief *causes* of acute vaginitis are :—(1) Traumatism, due to pins, peas, and worms in children, or in the adult an irritant pessary, or other foreign body (such as a letter), too powerful injections, or excessive coitus ; (2) Gonorrhœa, which is hard to diagnose from non-specific acute vaginitis excepting by its severity and history ; and (3) Spread from adjacent parts. A severe acute vaginitis is probably of gonorrhœal origin<sup>2</sup> and the danger of this rests in the liability to endometritis, pyosalpinx, peri- or parametritis, cystitis and ascending pyelitis. The *treatment* consists of rest, saline purges with hyoscyamus to allay the pain, copious warm drinks, hot hip-baths, and douches of weak carbolic,

<sup>1</sup> *The Lancet*, August 1, 1896.

<sup>2</sup> The *b. coli*, and possibly other pyogenic infections, may also cause acute vaginitis.

Condy (pot. permang. gr. x. to the pint), or corrosive sublimate, and after a few days some astringent lotion such as sulpho-carbolate of zinc (2 drs. to the pint), glycerine of subacetate of lead (4 drs. to the pint), alum, or tannic acid (1 dr. to the pint).

β. In CHRONIC VAGINITIS there is a thick continuous *opaque white* discharge with local signs of inflammation. The *causes* are (1) antecedent acute vaginitis; (2) various constitutional conditions, such as general debility, strumous (*i.e.*, tuberculous) diathesis, diabetes, old age, alcoholism, anæmia, syphilis, rheumatism, and convalescence from fevers; (3) new growths in the vaginal walls, such as epithelioma, condyloma, etc.; (4) irritant foreign bodies and other causes mentioned under acute vaginitis. The *treatment* consists of tonics and other remedies for any constitutional disease present; combined with warm douches (100° F.), containing sulphate of zinc or sulphate of copper, or weak chloride of zinc, or the remedies mentioned under acute vaginitis. Local applications are made with Ferguson's speculum, and a cotton swab dipped in silver nitrate solution (5 per cent.) or corrosive sublimate (1—1,000), especially in gonorrhœal cases.

(b) LEUCORRHŒA OF UTERINE ORIGIN may be due to endocervicitis, endometritis, subinvolution (see Menorrhagia), fibroids or polypi of the uterus (see Menorrhagia), cancer of the uterus (see Menorrhagia), concurrent peri- or parametritis (see Pelvic pain), and lastly, to various constitutional causes such as the gouty, rheumatic, anæmic, syphilitic, or tubercular diatheses.

I. In ENDOCERVICITIS, or inflammation of the cervix, the discharge is more or less constant, and consists of *glairy material* like white of egg. The other symptoms are:—(1) the cervix is swollen, hard, and may present retention cysts; (2) perhaps presenting catarrhal patches which bleed readily on touching; and (3) menorrhagia or dysmenorrhœa is frequently present. Endocervicitis may have to be *diagnosed* from *cancer* of the cervix. Here the age is not much guide, as cancer of the cervix may come on in a patient as young as 26. In cancer there is a shorter history and it soon breaks down, is friable to the touch, and there is often a slightly blood-stained discharge. Microscopic examination of scrapings will aid the diagnosis. When fixity of

the uterus and cachexia appear in due course, the diagnosis is simple. *Causes and Treatment* below.

II. In ENDOMETRITIS, or inflammation of the body of the uterus, the discharge comes in gushes when the patient rises or walks about ; and it has usually a *curdy character*. Endometritis is generally accompanied by both menorrhagia and dysmenorrhœa, and is often associated with displacements of the uterus. Bimanually the uterus is slightly enlarged and tender, and on passing the sound the interior is tender to touch, it readily bleeds and irregularities of the surface may be felt. Sometimes there is a history of recurring abortions or of sterility.

*Causes* of endocervicitis and endometritis :—(1) Cold during menstruation ; (2) extension upwards of vaginitis ; (3) retained fœtal products after labour or abortion ; (4) subinvolution ; (5) flexions and versions ; (6) instruments causing local injury or sepsis ; (7) intra-uterine growths ; (8) fevers and various blood states, such as the rheumatic diathesis ; and finally, it may come on insidiously without any definite cause, especially in old age (senile endometritis).

*Diagnosis.* Occurring in middle or advanced life endometritis and endocervicitis may have to be diagnosed from *cancer of the body* of the uterus by its more prolonged history, by there being less cachexia, and by the relative absence of those three pathognomonic features of cancer—pain, profuse hæmorrhages, and offensive discharge. Of all the local signs of cancer, fixity of the uterus is the most certain ; and the diagnosis may be aided by curetting and examining with the microscope.

*Treatment.* Both diseases call for a certain amount of hygienic and general treatment, especially if there has been much menorrhagia. Local douches and astringents are of very little use, and there are practically two methods of local treatment :—(1) the application of strong carbolic or other corrosive to the interior by means of Playfair's probes ; (2) dilating the cervix and curetting the interior. Endocervicitis is treated by applications of silver nitrate or copper sulphate (gr. xl— $\bar{5}$ j) applied by the use of a Playfair's probe, passed through a Ferguson speculum to protect the vaginal wall. Hot douches are used twice or thrice daily (never less than a quart at a time) ; and tampons of ichthyol

(10 per cent. in glycerine) are inserted after the douches or applications. Constipation must be avoided.

§ 322. **Dysmenorrhœa** is pain during the menstrual period. There are three varieties—(I.) NEURALGIC or SPASMODIC DA., in which the pain is paroxysmal, situated chiefly in the hypogastrium, begins a few hours before the flow and lasts for one to two days; (II.) INFLAMMATORY DA., in which the pain is dull, aching, persistent, situated sometimes in the small of the back and down the legs, begins several days before the flow, and is relieved by the flow, especially when it is profuse; and (III.) MEMBRANOUS DA., in which the pain is severe, paroxysmal, and relieved as soon as the membrane is passed. In the *first* named, local examination reveals nothing wrong in the uterus or its appendages, and the menstrual flow is usually natural. In the *second* variety, examination generally reveals some abnormality in the uterus or its appendages, *e.g.*, endometritis, fibroids, adhesive bands, ovaritis; and it not infrequently dates from a particular time, *e.g.*, a confinement or abortion. The *third* is distinguished by the passage of a membrane, which is distinguished from that of abortion by its being passed every month.

*Causes.* The causes of neuralgic dysmenorrhœa are those of a general character which lead to neuralgia in other parts. The causes of varieties II. and III. are those which lead to inflammation of the uterus or its appendages. All three varieties have been variously ascribed to obstruction of the flow by flexions of the uterus, or by constriction of the cervix, or to the undue excitation of uterine contractions.

*Treatment.* I. The neuralgic form usually calls for general treatment, hygienic, dietetic, and tonic. Treatment directed to the diathesis, as in rheumatic persons, may effect a cure. It is very important in this and in the other varieties to avoid constipation. Warm baths, and especially Turkish baths, are very valuable in my experience. Remedial treatment at the time of the period consists of hot bottles to the hypogastrium, hot drinks, feet in hot water, cannabis indica, belladonna, camphor, sal volatile, bromides, castor, antipyrin, and morphia (with great caution). Dr. Champneys recommends tr. castoreum (℥ xx.).



II. The inflammatory form admits of the same symptomatic treatment as the foregoing. The remedial treatment should be directed to the inflammatory lesion which is the causal agent. Depletory methods are indicated. Very hot douches (110° F. for 10 minutes) should be given twice daily. In severe cases removal of the appendages has been adopted.

III. For the membranous form the symptomatic treatment is as above, with the subsequent dilatation and curetting of the interior during the interval. In all three forms division of the cervix often relieves the condition, a fact which supports the idea of it being not infrequently due to flexion at, or stenosis of, the inner os.

§ 323. **Menorrhagia and Metrorrhagia.** The first of these terms indicates an excessive flow at the monthly period; the second indicates irregular hæmorrhage from the uterus, irrespective of the period. It is difficult to separate these two symptoms, as their causes are more or less identical and they very often occur together.

Hæmorrhage of this kind arising in women *under* 30 may be due to the following causes in order of frequency:—Endometritis, Constitutional conditions, Fibroids and Polypi of the uterus, Pelvic inflammations, Subinvolution of the uterus, Retroverted uterus incarcerated in Douglas' pouch, Ovarian tumours (occasionally), Inversion of the uterus, and Extra-uterine fœtation. Flexions and versions of the uterus are said to cause no symptoms unless attended by pelvic inflammation or adhesions.

In women *over* 30 the above causes also may give rise to hæmorrhage, but in addition there are two other conditions which may be causally associated, viz. (1) the Menopause and (2) Malignant Disease. The sudden supervention of *metrorrhagia* with *acute pain* should always suggest a miscarriage or an extra-uterine fœtation (§ 330) to one's mind.

In women *past the menopause* some gross lesion of the uterus, especially Cancer or Uterine Fibroid, is nearly always in operation.

Many of the above conditions are elsewhere dealt with, but menorrhagia is the chief symptom referable to the reproductive organs in:—(I.) Certain Constitutional conditions; (II.) Uterine

Fibroid or Polypus; (III.) Subinvolution (in persons under 30); (IV.) the Menopause; and (V.) Malignant disease (in persons over 30). These conditions will therefore be differentiated here.

§ 324. Menorrhagia may, in the first place, depend upon certain CONSTITUTIONAL CONDITIONS. (1) Certain girls of a plethoric habit of body, usually with florid countenances, may be troubled with too profuse periods all their lives, and a tendency to excessive flow on any trivial exciting cause. (2) Prolonged lactation or too many and too frequent pregnancies; (3) residence in tropical climates; and (4) mental over-work, especially if combined with a sedentary life, are said to produce it. (5) The vague condition we call hysteria, especially in that variety which is subject to flush storms, is frequently attended by menorrhagia. (6) The back pressure in the circulation which attends some heart and liver diseases finds more or less relief in this way. The differential characters of menorrhagia due to these causes are:— (1) The menstruation may occur every third or second week, or even weekly, though in point of quantity it may or may not be increased. The flow, moreover, may be very readily excited, as by a hot bath, or after a day of unusual exercise. (2) The general symptoms after a time point to anæmia, combined with the symptoms of the constitutional cause in operation.

§ 325. Menorrhagia may, secondly, be due to a UTERINE FIBROID. The symptoms vary with the position of the tumour. These tumours may be submucous, interstitial, or subserous. When the fibroid is submucous or interstitial, the symptoms of uterine fibroid are (1) menorrhagia and metrorrhagia. (2) Leucorrhœa and sometimes dysmenorrhœa are present. (3) On examination with the sound the uterine cavity is found to be enlarged; and (4) on bimanual examination a tumour or enlargement of the uterus can be detected. There is a tendency for the submucous variety to become polypoid, remaining attached to the uterus by a pedicle. The *subserous* fibroid may present no symptoms at all for many years, and may even then be discovered by accident. Amenorrhœa may accompany such cases quite as often as menorrhagia, and the latter is never profuse. In short, pressure symptoms may be the earliest indication of a subserous fibroid. In uterine fibroids of all kinds the rate of growth, though

it varies somewhat, is nearly always very slow; but as the tumour increases we get symptoms of pressure upon the surrounding organs, such as frequent micturition, varicose veins, neuralgia in legs and back, indigestion, difficult respiration, or hydronephrosis.

UTERINE POLYPUS is another cause of menorrhagia. They are of three kinds. The most common forms are (1) fibroid polypi (which result from submucous fibroids), and (2) mucous polypi. (3) Placental and fibrinous polypi occur, the first after labour or abortion, arising from retained portions of the placenta, the second from the stump of a growth previously removed.

Polypi present at first most of the symptoms of submucous fibroid. When very small they can be made out with certainty only by dilating the os and exploring the interior. Later on the polypus may be seen hanging from the os into the vagina on examination with the speculum. After a time it may slough, and cause an offensive discharge.

§ 326. SUBINVOLUTION, or the non-return of the uterus to its normal size, is a very frequent cause of menorrhagia after labour or abortion. After a confinement the uterus immediately begins to diminish in size, and at the end of about two months it resumes its normal size of  $2\frac{1}{2}$  inches. In cases of subinvolution we find (1) on the passage of a sound that the uterus is enlarged; (2) it tends in most cases to be retroverted and lower than normal. (3) The patient generally complains of backache, bearing-down pain, and leucorrhœa; and (4) lassitude, weakness, and general malaise are usually present.

The *causes* of subinvolution are important. (1) Getting up too soon after childbirth is probably the reason why this condition occurs so frequently among the poor. (2) Retained membranes or portions of placenta; (3) pelvic inflammation; (4) delayed labour or over-distension of the uterus; and (5) the practice of not suckling the infant, account for the condition frequently enough; and therefore it is more often met with in those who have had numerous and rapid pregnancies.

§ 327. THE MENOPAUSE (cause V. of Menorrhagia), or climacteric, is the epoch at which the sexual activity of the female undergoes involution, when the menses, which are the sign of that activity, cease. This cessation of the menses may take place in three

ways: (a) they may cease gradually, and generally more or less irregularly; (b) quite suddenly; (c) they may be attended by a series of hæmorrhages. The last-named method, which is quite as frequent as either of the other two, is the one with which we are now concerned.

The existence of this cause of menorrhagia or metrorrhagia can only be recognised by the attendant phenomena. (1) The age of the patient varies considerably between 35 and 55, the average being about 45. (2) The occurrence of "flush storms," which consist of a hot stage, a cold stage, with or without shivering, and sometimes a stage of perspiration. (3) The other nervous phenomena which may occur at this time are extremely varied. There is generally an irritability and restlessness, and generally also a marked tendency to depression of spirits, and to burst into tears at the slightest provocation. This may amount, especially when there is mental heredity, to definite melancholia. Sexual perversions, with a marked tendency to excess of all kinds, are apt to occur. (4) While fibroids and other gross lesions may undergo involution at this epoch, carcinoma, if there be a predisposition, may sometimes make its appearance, and the case should be carefully watched from this point of view.

§ 328. MALIGNANT DISEASE of the uterus (cause VI. of Menorrhagia) is clinically met with in four forms:—(a) Cancer of the cervix, chiefly met with in multiparæ, between the ages of 25 and 70; (b) cancer of the body, which is chiefly met with in sterile women, between the ages of 50 and 60; (c) sarcoma of the uterus, which is rare, unless we include under that term certain fibroids which appear to take on the malignant features of spindle-celled or large round-celled sarcoma; and (d) deciduoma malignum, a very rare form following parturition.

The symptoms differ in the first three varieties. (a) CANCER OF THE CERVIX consists of an epitheliomatous growth which usually runs a somewhat rapid progress. (1) On digital examination the os has a friable granular feel which is so characteristic that this feature and the blood-stained discharge upon the finger are alone, in experienced hands, sufficient to diagnose the disease. (2) In some cases examination reveals a mushroom-like growth ("cauliflower excrescence") hanging down into the vagina, readily



breaking down and readily bleeding. It has a tendency to spread to the vaginal wall, to the utero-sacral ligaments, broad ligaments, and body of the uterus, leading to a fixity of the uterus and hardness which is easily made out on palpation. (3) Metrorrhagia and menorrhagia are present. (4) In the intervals between the marked hæmorrhages there is a continuous leucorrhœa of pinkish-brown colour, often of a very offensive odour. (5) Local pain is usually a late symptom, but, like the wasting and the cachexia, is sure to supervene sooner or later.

(b) CANCER OF THE BODY of the uterus is chiefly met with in sterile women over 50 years of age. The symptoms are—(1) Metrorrhagia, and in the intervals pinkish brain-like matter is discharged, which has a very offensive odour. (2) Pain is an early symptom, and very severe. (3) On bimanual examination the uterus is found to be enlarged. Later on, as the disease extends to the broad ligaments, the uterus becomes fixed. This fixity to the educated finger is very characteristic of the disease. On the passage of a sound considerable hæmorrhage may take place. (4) The cachexia and other general symptoms resemble those of cancer elsewhere.

(c) SARCOMA OF UTERUS is a relatively rare condition. Its symptoms do not differ materially from those of uterine fibroid, excepting in the rapidity with which the case progresses, and the liability to deposits elsewhere.

*Prognosis of Menorrhagia.* Menorrhagia of itself is not fatal to life, but some forms are very intractable, and lead to considerable anæmia, debility, discomfort, and inability to fulfil the duties of life. (1) The menorrhagia of the MENOPAUSE and of SUBINVOLUTION tends to spontaneous recovery, and that which is due to CONSTITUTIONAL conditions is usually amenable to treatment; so also in many cases is that due to PELVIC INFLAMMATION. (2) ENDOMETRITIS is perhaps one of the most intractable of the causes, though this also is remediable by local treatment. (3) The prognosis in a case of FIBROID tumour depends very much upon its position. The submucous varieties (and mucous polypi, § 325) are readily treated, but if neglected these may slough and produce death by exhaustion and septic intoxication. The subserous form is next in order of gravity, though this may

give but little trouble for a great many years. The interstitial form is the most serious, and if there be much loss of blood and consequent prostration the patient can only live a life of invalidism. When of large size these tumours are very difficult to treat. Fibroids, when occurring near the menopause, should not be treated surgically, because they may spontaneously disappear. If the patient be not near the climacteric, and if the symptoms both general and local are serious, the question of extirpation of the uterus is to be considered; and it is now done with comparative safety. Dr. Gow<sup>1</sup> has published some very successful statistics of the operation, which compare very favourably with those of ovariectomy and other major operations.

(4) CANCER is the most serious of all the causes of menorrhagia, and if untreated tends in the course of one to two years to terminate in death from exhaustion, hæmorrhage, peritonitis, or uræmia by involvement of the ureters. The chance of recovery depends upon the disease being discovered and treated surgically *at an early stage*. If cancer of the cervix is discovered before it has spread to the parts around, or if cancer of the body is taken in hand while the uterus is still freely movable, amputation of the one or extirpation of the other offers a fair prospect of recovery.

*Treatment of Menorrhagia.* (a) Symptomatic, in all forms. To relieve the hæmorrhage calcium chloride in large doses (20 grains or more) is the most recent and most popular of remedies, as it promotes the coagulability of the blood. Ergot, acid. sulph. dil., tinct. hyrastis, tinct. hamamelis, hot douches, tonics, chloride of iron, quinine, nux vomica, are all useful. If the hæmorrhage is alarming (*e.g.*, in a large fibroid) the uterus must be plugged with iodoform gauze after dilating the cervix up to a No. 7 Hegar dilator (§ 318). Leave the plug in for twenty-four hours only, then explore the interior. (b) Remedial treatment is directed to the cause, and must be adopted in addition to the foregoing. (c) In all cases general measures are required—the food must be nourishing, exercise must be avoided near the period, and the patient must rest in bed while the flow is profuse. While strong purgatives on the one hand must be avoided, it is extremely

<sup>1</sup> *Lancet*, December, 1899, Med. Soc. Report.

important on the other hand to avoid constipation. For the menorrhagia of the menopause bromides and calcium chloride are the best remedies, and these also relieve the attendant discomforts.

§ 329. **Amenorrhœa** is that condition in which the catamenia are either deficient or absent. The term *primary* amenorrhœa is applied to the condition in which menstruation has never occurred, as in rare cases where the patient is of a masculine type, where there is a congenital absence of the organs concerned in the function, and also in cases of infantile uterus and undeveloped ovaries. *Apparent* amenorrhœa is that form in which there is a feeling of fulness in the breasts and abdomen every month, but the menstrual flow is restrained behind an imperforate hymen, an occluded os or vagina. In *secondary* amenorrhœa the flow, after having been once established, ceases or becomes deficient for a time. *Physiological* amenorrhœa is the cessation of the menses flow which occurs in pregnancy, a fact which must always be borne in mind even amongst the most irreproachable patients.

In PREGNANCY, the physiological cause of amenorrhœa, (a) the *general symptoms* are as follows:—(1) morning sickness is usually one of the earliest, coming on about the 1st, or 2nd, and ceasing at the 4th month; (2) the mammæ present a dark areola around the nipple, they become enlarged, and after the 3rd month contain milk. (b) The *local signs* are—(1) on digital examination there is a *softness* of the os which is unmistakable to the educated finger; (2) a gradual increase in the bulk of the uterus commences at once. The foregoing are the earlier symptoms; and about the 3rd or 4th month we have a series of unmistakable signs, viz., (3) about the 18th week fœtal movements can be felt by the physician, and (4) also about the 18th week the fœtal heart sounds (at the rate of 120 to 150 a minute) can be heard on auscultation, usually midway between the umbilicus and left anterior superior spine, and (5) ballottement can be made out about the 5th or 6th month.

The *causes* of SECONDARY AMENORRHOEA may be divided into constitutional and local causes. (a) *Constitutional* causes are by far the most frequent, and undoubtedly the most common of these are anemia, or chlorosis and phthisis. It also occurs after severe

illness, on account of some great grief (mental shock), and during prolonged lactation. (b) The most important of the *local* causes is perhaps an ovarian tumour, in which the state of the catamenia varies, but usually the flow is absent or irregular. Other causes are a chill during menstruation, inflammatory conditions in the pelvis, superinvolution of the uterus, and extra-uterine foetation.

*Treatment* in constitutional causes consists in taking plenty of fresh air, exercise, good food, and general healthy living combined with iron tonics. Warm baths, especially warm hip-baths at the expected time, are useful. It is very important to keep the bowels regularly acting, and the old-fashioned remedy of the aloes and iron pill is most beneficial. Permanganate of potash in 2 gr. pills has been recommended. It is always advisable to adopt the tonic treatment in young unmarried girls, and it is only after these have failed that local causes should be suspected, or at any rate locally investigated (compare § 318).

SUDDEN SUPPRESSION of the catamenia is a form of amenorrhoea which requires special treatment. The flow has probably come on normally, and then suddenly ceased on the 2nd or 3rd day, and the patient suffers a good deal of general discomfort. In such cases the feet should be put in hot water or a mustard bath or a warm hip-bath, and the patient placed in a thoroughly warm bed with hot bottles and given hot drinks. Subsequently saline purgatives in constant small doses, and general attention to the health are indicated. When the time of the expected period again comes round the procedure just mentioned should be adopted.

§ 330. **Extra-uterine pregnancy** (or foetation), may become manifest by menorrhagia, metrorrhagia, or amenorrhoea. The term is applied to the condition where pregnancy takes place outside the uterus, generally in the Fallopian tube.<sup>1</sup> The tube usually ruptures at the 2nd or 3rd month after fertilisation, either into the broad ligament (extra-peritoneally) or into the peritoneal cavity.

*Symptoms.* (1) In many cases paroxysmal pains are experienced in one iliac fossa; (2) there is a history of amenorrhoea for some weeks or a month or two over time; followed in most cases by a history of irregular hæmorrhages from the uterus. A membrane or cast may be discharged from the interior of the uterus at the same time. (3) Other symptoms of early pregnancy, such as morning sickness, are present. (4) On bimanual examination a swelling is found in the fornix. In most cases, however, none of the above symptoms may be noticed by the patient, and advice may not be sought until the time of rupture of the tube, when the patient consults us for *severe pain* and *hæmorrhage*. Extra-peritoneal rupture is attended and followed by the symptoms of pelvic hæmatocoele (§ 335);

<sup>1</sup> The question of ovarian pregnancy (*i.e.*, fecundation of the ovum *in situ*) is still *sub judice*.



intra-peritoneal rupture by the symptoms of perforative peritonitis (§ 170). If the rupture takes place about the fourth week the shock is not so severe, and the hæmatocele often remains extra-peritoneal.

*Causes.* It usually occurs in women over 30, who have been sterile, or have had no children for several years. Mr. Lawson Tait held the theory that after salpingitis the cilia of the tube were destroyed, and so led to fertilisation taking place in the tube instead of in the uterus.

*Diagnosis.* The diagnosis is often extremely difficult, and as above mentioned, the patient may complain of no symptoms until rupture occurs. The symptoms which should be inquired for are the sudden amenorrhœa, early pregnancy symptoms, irregular hæmorrhages, and pains in one iliac fossa. If, with these symptoms, the os uteri is found to be patulous and soft, and the uterus slightly enlarged, it is probable that the case is one of extra-uterine pregnancy.

*Prognosis.* The condition is extremely serious; 70 per cent. die if untreated. (a) An *extra-peritoneal* rupture is not so immediately serious, since bleeding is checked in the limited space formed by the layers of the broad ligament. If untouched, in favourable cases, the foetus may even live till full term, the patient going through a spurious labour; after which the liq. amnii and placenta are absorbed, and the foetus becomes mummified, remaining *in situ* without causing any symptoms, and only discovered accidentally on *post-mortem* examination. (b) In less favourable (though, unfortunately more common) cases, rupture into the abdominal cavity takes place (*intra-peritoneally*) giving rise to alarming and even fatal hæmorrhage. In rare cases the foetus may live in the peritoneum till full term, and be delivered by abdominal section. In some cases of variety (a) a *secondary* rupture into the peritoneum occurs, and the case then runs the course described in (b).

*Treatment.* The treatment of extra-uterine pregnancy may be divided into three stages: (1) If diagnosed before rupture occurs laparotomy must be performed and the tube removed; (2) if diagnosed after intra-peritoneal rupture the same procedure must be adopted; (3) if an extra-peritoneal rupture has occurred, treat with rest, as in hæmatocele.

§ 331. **Pelvic pain.** Pain in and about the pelvis is perhaps one of the commonest symptoms of disorder of the female reproductive organs. "Bearing down" is often spoken of; and "back-ache" or pain over the sacrum, is so constant a feature of uterine disorders that it has come to have that association in the minds of the laity. The position and character of pelvic pain vary with the different maladies, but its degree is largely influenced by the temperament of the patient. Reference has already been made to painful menstrual periods (dysmenorrhœa), but the causes of a continuous pain (without reference to the menstrual period), such as that now in question, may be conveniently grouped into (a) those pains which come on more or less suddenly (acute conditions), and (b) those which come on more or less insidiously (chronic

conditions). It must however be remembered that no hard and fast rule can be laid down in this respect.

a. *The pelvic pain came on acutely and recently; it is accompanied by more or less* CONSTITUTIONAL DISTURBANCE—PERI- or PARAMETRITIS, INFLAMMATION of the UTERINE APPENDAGES, PELVIC HEMATOCELE, ACUTE CYSTITIS, or some other INFLAMMATORY CONDITION within the pelvis, may be suspected, and the reader should first turn to § 332.

*If the PAIN has come on VERY SUDDENLY with FAINTNESS and NAUSEA—turn first to PELVIC HEMATOCELE, § 335; and if such a sudden pain be accompanied by METRORRHAGIA, it is suggestive of MISCARRIAGE, or EXTRA-UTERINE FŒTATION, § 330.*

§ 332. **Perimetritis (pelvic peritonitis)**, which is one of the most frequent causes of pain, is an inflammatory condition affecting the peritoneal surfaces around the uterus and its appendages. Exudation may be present, and in chronic cases the adhesions lead to a matting together of the pelvic viscera.

The *symptoms* of ACUTE PERIMETRITIS consist of (1) acute pain across the lower part of the abdomen; (2) the abdomen is distended and tender to palpation, and the patient lies on the back with legs drawn up; (3) on examination, the vagina is found to be extremely tender; (4) on vaginal examination 48 hours later the uterus is found to be fixed, with a certain amount of exudation surrounding it, which may be so great as to push the uterus forwards; (5) the general symptoms consist of high fever, quick pulse, with vomiting.

In CHRONIC PERIMETRITIS (1) the pain is felt across the lower part of the abdomen, and is often greater on one side; backache is usually present. The pain is constant, of a bearing down character, worse at the menstrual period, (2) dysmenorrhœa and sometimes symptoms of endometritis accompany it; (3) on examination the mobility of the uterus is found to be diminished, and thickenings, *chiefly in the posterior fornix*, can be felt behind the uterus, in which situation a kind of "roof" to the vagina exists. (4) The general symptoms consist of an inability to stand or to walk for any length of time; and in severe cases chronic invalidism with mental depression or hysteria results.

*Causes.* (1) Vaginitis or endometritis extending up by way of

the Fallopian tubes is a common cause of pelvic peritonitis; thus, after confinement or abortion, acute pelvic peritonitis is often caused by this extension of inflammation. (2) Menstrual regurgitation, or a chill during the menstrual period, may give rise to pelvic peritonitis. (3) Chronic pelvic peritonitis may be set up by ovarian tumours, fibroids, cancer, or tubercle. *Prognosis and Treatment* below.

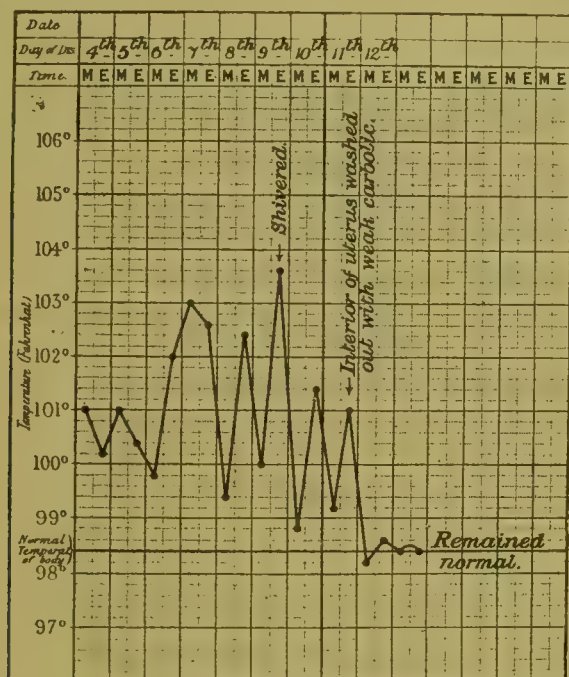


Fig. 92.—Chart of SEPTIC ABSORPTION showing effect of intra-uterine irrigation. Annie H., æt. 33, recently confined; septicæmia seemed to be threatening, but after thoroughly washing out the interior of the uterus, all the symptoms subsided.

§ 333. Parametritis (pelvic cellulitis), which may be another cause of pelvic pain, is an inflammation originating in the connective tissue of the pelvis adjacent to the uterus. This also may be acute or chronic.

The symptoms of ACUTE PARAMETRITIS consist of (1) intense pain across the lower part of the abdomen, usually *shooting down one leg*; and the patient usually lies with *one leg drawn up to relieve the pain*. In a few cases no pain is complained of at first. (2) On examination swelling and tenderness are made out in *one of the*

*lateral fornices*, or one postero-lateral quarter of the pelvis. No swelling is felt in Douglas' pouch (the posterior fornix) unless pelvic peritonitis is also present. (3) The general symptoms consist of fever, generally of a hectic type, with quick pulse; in those cases where no pain is felt attention is drawn to the condition by the rise of the patient's temperature.

In CHRONIC PARAMETRITIS there is backache, dysmenorrhœa, frequently uterine displacement (due to the contraction of the inflammatory tissue), and symptoms of endometritis. When it results in suppuration or "phlegmon" the pus may make its way in various directions upwards or downwards.

*Causes.* Parametritis usually follows labour or abortion in which injury to the cervix, vagina, or perineum has occurred, with consequent *entrance of septic matter*. Injury by septic instruments may also produce it; and thus clumsy attempts at procuring abortion form a possible source. Apart from these, parametritis is practically unknown, though some authors have described an idiopathic form. The serious results which may be produced by septic absorption and the prompt effect of thorough asepsis are well illustrated in Fig. 92.

*Course and Prognosis.* (a) In *acute perimetritis*, the acute symptoms should subside in a week; if widespread adhesions are present, part of the exudation will be absorbed, and part will remain, giving rise to the symptoms of chronic perimetritis. *Chronic perimetritis* tends to be incurable. The prognosis will depend (i.) upon the extent of the inflammation, and (ii.) its cause. If it is the sequel to an acute attack with widespread adhesions the patient will probably have chronic pelvic pain and dysmenorrhœa all her life. If due to extension from a diseased organ, the patient will be subject to relapses with acute pain after any imprudence in the way of chills or over-exertion.

(b) In *acute parametritis*, if treated properly, the fever should subside in a week; and the exudation will probably be absorbed in 3 weeks. If the fever continues for 4 or 5 weeks pus has formed, and the patient will be invalided until the pus finds an exit (which may not be for months). The swelling felt in one lateral fornix becomes larger, pushing the uterus to one side, and later on a firm lump, which may extend to the iliac fossa, is felt along Poupart's



ligament. The pus may point in the iliac fossa or, following the line of the vessels, in Scarpa's triangle; or it may burst into the vagina, bladder, or rectum. In *chronic pelvic cellulitis*, adhesions and fibrous tissue are formed, rather than pus. These may be absorbed in time, but flexion or version of the uterus is a common result of the contraction of the utero-sacral ligaments which occurs in parametritis.

*Treatment.* *Acute* peri- and parametritis must be treated by (i.) absolute rest in bed; (ii.) hot fomentations, turpentine stupes to the abdomen, and hot vaginal douches; (iii.) saline purges; (iv.) morphia, if necessary, to alleviate the pain. Quinine 3 grs. t.d. in milk may be given. *Preventive* treatment consists especially (1) in cleanliness of the hands of the nurse or doctor who attends a case of labour or abortion, and (2) in the curing of a vaginitis or an endometritis before it can extend up to the Fallopian tubes. The treatment of *chronic* peri- and parametritis consists of (1) the administration of hot vaginal douches (up to 120° F.) daily, each douche lasting 10 minutes; (2) ichthyol tampons; (3) treating the pain, dysmenorrhœa, menorrhagia, and other symptoms as described under those conditions. Cold or damp and undue exertion in walking or standing must be avoided; and a certain daily interval of rest in the recumbent position should be ordered. During and after convalescence, it is highly important to avoid constipation; aperients or enemata should be given. When suppuration has occurred, the pus must be evacuated by surgical procedure either by trochar or free incision, preferably per vaginam.

§ 334. **Inflammation of the Uterine Appendages** (viz., Ovaritis and Salpingitis) may also be a cause of pelvic pain.

OVARITIS is inflammation of the ovary, and it should be distinguished from ovarian neuralgia. The *symptoms* of ovaritis are so frequently accompanied by those of perimetritis that it is difficult to differentiate them. Indeed *acute ovaritis* is found solely with acute peri- or parametritis (*q.v.*). *Chronic ovaritis* may be recognised by—(1) severe pain at the pelvic brim, extending down the thigh of the affected side; (2) pain increased by any pressure on the pelvic viscera (*e.g.*, by much standing, constipation, or flatus in the abdomen, and in severe cases by sitting);

(3) menorrhagia and dysmenorrhœa, because endometritis so often accompanies ovaritis; and (4) dyspareunia. (5) The ovary is usually prolapsed and therefore, per vaginam, a swelling, the size of a walnut, is found at the site of the ovary, to one side of or behind the uterus, acutely tender to touch, which causes a sickening pain. *General symptoms*, referable for the most part to the nervous system, very frequently supervene. The *causes* of (1) acute ovaritis are sepsis after labour, abortion, or surgical operations; (2) chronic ovaritis may be due to the same causes as perimetritis, to alcoholism, to certain fevers (*e.g.*, mumps), or to the suppression of menstruation by a chill.

**SALPINGITIS** (inflammation of the Fallopian tubes) occurs in 3 forms, hydro-, pyo-, and hæmato-salpinx. (i.) When the fimbriated end of the tube is closed by adhesions, the exudation within, unable to escape, tends to accumulate in the tube instead of escaping by the uterine opening (hydro-salpinx); (ii.) when the tubes are filled with pus (tubercular or gonorrhœal) the condition is named pyosalpinx; (iii.) in hæmatosalpinx the tubes are filled with blood.

The *symptoms of salpingitis* are (1) pain across the lower part of the abdomen, usually greater on one side, often shooting down one leg; (2) on examination a sausage-shaped swelling is found, usually double, running from the lateral fornices to Douglas' pouch; (3) as perimetritis usually accompanies it, the uterus is less mobile than normal; (4) dysmenorrhœa and menorrhagia are usually marked. (5) As regards the general symptoms—in hydrosalpinx there may be none, but pyosalpinx is accompanied by fever. In a pyosalpinx of sudden onset (gonorrhœal) the fever may be very high. *Causes.* (1) Acute salpingitis is due to septic or to gonorrhœal infection extending upwards; (2) the chronic pyosalpinx, which is found more commonly in young women, is usually due to tubercle; (3) a chronic or subacute vaginitis or endometritis extending upwards may result in salpingitis; (4) hæmatosalpinx is due usually to a ruptured extra-uterine pregnancy.

The *prognosis of ovaritis* depends on the extent of the inflammation around. If there is much matting the case is really one of perimetritis. If the inflammation is confined to the ovary the prognosis is favourable, provided the cause be removable and the patient is not of a neurotic constitution. In *salpingitis*, sterility may result from adhesions closing the fimbriated extremity (though this cannot be diagnosed with certainty). Pyosalpinx is dangerous to life, as it may at any time burst into the peritoneum. Tubercular salpingitis is very chronic, and less painful than the other forms. In all forms there is a tendency to relapse, and to peritonitis by extension rather than to spontaneous cure.

*Treatment.* Acute and chronic ovaritis are treated like perimetritis (*q.v.*), together with hot applications to the hypogastrium when the pain is severe. Blisters and iodine applications over the iliac region have been recommended. If the suffering is severe the ovaries may require to be removed. Constitutional treatment must not be neglected—bromides, pot. iod., and tincture of belladonna are beneficial. In *acute salpingitis*,

where the condition can be certainly diagnosed, laparotomy should be performed and the tube removed. In other cases rest in bed with hot douches may tide over the acute stage. In *chronic* salpingitis rest, hot douches and the ichthyol tampons may be tried for a period of 2 years at least. If this treatment fail it will probably be necessary to remove the tubes.

§ 335. **Pelvic hæmatocele** is an effusion of blood either into the peritoneal cavity (intraperitoneal) or into the connective tissue of the broad ligament (extraperitoneal). Here there is a very *sudden onset* of (1) severe pain in the lower part of the abdomen, accompanied by (2) faintness, perhaps unconsciousness; with (3) nausea, and in some cases vomiting. (4) If the hæmatocele occurs during the menstrual period the flow ceases usually for a few hours at the onset of the pain, but may return again. (5) On examination, the uterus, in the intraperitoneal variety of pelvic hæmatocele, is found pushed forwards behind the pubes, while in the extraperitoneal variety the swelling is smaller, and causes a lateral displacement of the uterus as in pelvic cellulitis. The intraperitoneal variety, if large, forms a lump which can be felt, on bimanual examination, both in Douglas' pouch and above the pubes, and the abdomen is distended and tender. After 48 hours, adhesions form and the uterus is fixed; and other signs of pelvic peritonitis may then ensue. As regards the general symptoms, the temperature begins to rise in 24 hours after the onset of pain, that is to say, when the pelvic peritonitis commences. *Diagnosis.* Intraperitoneal hæmatocele is diagnosed by the sudden onset of pain, the local signs, a history of suddenly suppressed menstruation, or a history pointing to extra-uterine pregnancy. The extraperitoneal hæmatocele is extremely difficult to diagnose from pelvic cellulitis, unless a history pointing to extra-uterine foetation be present.

*Causes.* (i.) Pelvic hæmatocele occurs mostly in multiparæ, between the ages of 25 and 35; (ii.) a reflux of blood during menstruation, variously ascribed to violent exercise, fright, or cold, may give rise to pelvic hæmatocele; (iii.) a ruptured ovarian cyst; (iv.) extra-uterine pregnancy (*q.v.*) is the usual cause of extraperitoneal hæmatocele, but this also may lead to intraperitoneal hæmatocele, when the tension bursts the broad ligament, and the blood enters the peritoneal cavity.

*Prognosis.* If hæmorrhage be large death has been known to occur in about an hour. In smaller hæmorrhages adhesions due to pelvic peritonitis or cellulitis follow, and the exudation may be (i.) entirely absorbed, or (ii.) may go on to suppuration with a danger of general peritonitis. The prognosis in such cases is that of pelvic peritonitis or cellulitis (*q.v.*).

*Treatment.* The collapse is treated by stimulants, the pain with morphia and other usual methods. The radical treatment depends upon the cause—(1) in the early stages, if there is no history or sign pointing to extra-uterine pregnancy, leave the patient at rest in bed with ice to the hypogastrium; (2) if there is a history of extra-uterine pregnancy laparotomy must be immediately performed. In later stages leave the tumour alone unless there is definite evidence of suppuration, when the case becomes one of pelvic abscess, and is treated as such.

b. *The pain is of a chronic character and of some duration, and is UNATTENDED by PYREXIA*—almost any of the different

diseases mentioned in this chapter may be suspected. Perhaps a vaginal examination may reveal ENDOMETRITIS, CHRONIC PERI- or PARAMETRITIS, or a UTERINE DISPLACEMENT; or careful bi-manual examination may reveal a PROLAPSED OVARY or an INFLAMED TUBE: Uterine Displacements and Pelvic Tumours alone remain to be considered.

§ 336. **Uterine Flexions and Versions.** The normal position of the uterus is one of slight ante flexion. The uterus undergoes physiological displacements according to the fulness of the bladder and rectum. In itself a displacement leads to no symptom; the symptoms so often associated with displacement are due in the majority of cases to the inflammatory processes in or near the uterus which have caused the displacement.

FORWARD DISPLACEMENTS (ANTEFLEXION). On examination bi-manually the os is found to be high up, and the fundus is felt unduly far forward. The sound passes with some difficulty. In single women a stenosis of the os or an elongated cervix may accompany a forward displacement of congenital origin. As above stated, *symptoms* may be entirely absent, and attention is first drawn to the condition when other mischief, such as pelvic inflammation, endometritis, parametritis, or a history of dysmenorrhœa, sterility, or constantly recurring abortions is present.

*Causes.* (1) A congenitally ill-developed uterus is often displaced forwards. A forward displacement is diagnosed to be pathological in origin, as distinct from physiological, by the lessened mobility of the uterus, and the pain set up on attempting to move it. Forward displacements are found in association with (2) pelvic peritonitic adhesions, and (3) cellulitis affecting chiefly the utero-sacral ligaments.

*Prognosis.* Ante flexion is a frequent cause of sterility. Its treatment is extremely troublesome, but if consistently and carefully carried out a radical cure is certainly to be expected unless the condition is due to a considerable degree of pelvic peritonitis or cellulitis, when the prognosis depends upon the removability of these conditions.

*Treatment.* Treatment must be directed to any pelvic peritonitis or cellulitis present (*q.v.*). Ichthyol tampons and hot douches



with purgative treatment will work wonders in the slighter forms. Massage is highly recommended where the ante flexion is due to the contraction of the utero-sacral ligaments. Division of the cervix is indicated in some cases.

BACKWARD UTERINE DISPLACEMENTS consist of *retroversion* and *retroflexion*. In a backward displacement there is also a certain degree of descent of the uterus. Retro-displacements in themselves cause no symptoms; and in some instances the uterus is congenitally retroverted. On examination a lump is felt in the posterior fornix continuous with the cervix; the os looks forwards in version and is normally placed in flexion. The sound passes backwards, and the lump in the posterior fornix moves when the sound or the cervix is moved. *Symptoms* arise when pelvic adhesions are present, or when the displaced organ interferes with other organs in the vicinity. In such conditions, a retroverted uterus gives rise to (1) pain in the small of the back and the lower part of the abdomen of a bearing down character; (2) dysmenorrhœa and menorrhagia; (3) constipation and painful defæcation. (4) If pregnancy occur the sickness of the early months is excessive, and after the 4th month there may be retention of the urine, with dribbling.

*Diagnosis.* The diagnosis of a backward displacement is not difficult; but the diagnosis of the cause may be obscure. It is important first of all to determine whether the uterus is freely movable or not, as the prognosis and treatment differ.

*Causes.* The causes of backward displacement are:—(i.) congenital; (ii.) the dragging of adhesions consequent on pelvic peritonitis; (iii.) changes in the uterine tissues, such as subinvolution, or tumours in the walls; (iv.) relaxation of the ligaments, as after pregnancy; (v.) sudden fall or strain; and in a few cases (vi.) a habitually overdistended bladder. Several of these causes may act in combination; thus, subinvolution together with a relaxation of the ligaments cause a retroversion with a certain amount of downward displacement of the uterus, as pointed out in Prolapse.

*Prognosis.* (1) So long as the uterus is freely movable and not enlarged, there may be no symptoms until pregnancy occurs, when, as pointed out above, the symptoms may become serious. Most

often, perhaps, constantly recurring abortions take place. (2) In the case where the uterus is bound down by adhesions there is a condition which, according to Playfair (*loc. cit.*), is "not fatal, but tends to life-long discomfort."

*Treatment.* (1) Where the uterus is freely movable replace it with a sound or by bimanual manipulation. A Hodge's pessary should be worn so long as the uterus gives any sign of returning to the backward displacement. Where there is pregnancy and the uterus cannot be replaced, it may be necessary to bring on abortion. Where the condition is due to adhesions it must be treated as in pelvic peritonitis and cellulitis.

§ 337. The following are some of the more important **Pelvic Tumours and Vaginal Swellings**. a. *Internal tumours*: (1) uterine fibroid; (2) cervical or uterine polypus; (3) cervical or uterine cancer; (4) retroverted uterus; (5) pelvic cellulitis; (6) ovarian tumour; (7) pyosalpinx; (8) appendicitis occasionally drops into the pelvis; (9) pelvic hæmatocele; (10) hydatid of the pelvis (a case of which is mentioned, § 181). b. *External swellings* or swellings about the vulva may be due to—(1) prolapse of the uterus; (2) inversion of the uterus; (3) prolapse of the vaginal walls (cystocele and rectocele); (4) cysts or tumours of the vaginal wall—*e.g.*, of Bartholini's gland; (5) uterine polypus with a long pedicle; (6) local conditions of the *vulva*, such as abscess or labial thrombosis, § 320; (7) *cysts of the vaginal wall* are usually found on the anterior wall, about the size of an egg, and painless.

Most of these various conditions have already been fully referred to, but three conditions which may appear as external swellings remain to be described—PROLAPSE OF THE VAGINAL WALLS, PROLAPSE OF THE UTERUS, and INVERSION OF THE UTERUS.

§ 338. **Prolapse of the vaginal walls** is very common in multiparae, especially of the anterior wall. It is then named cystocele, because of its close connection with the bladder; indeed, the anterior vaginal wall may draw down the posterior wall of the bladder along with it. Prolapse of the posterior wall may occur, and when the rectum is prolapsed along with it is named rectocele. But as the rectum is not so intimately attached to the posterior vaginal wall a prolapse of that wall is not usually a rectocele. The only symptom in addition to the swelling may be difficulty in passing water until the prolapsed part is pushed up. The diagnosis from a cyst of the vaginal wall is made by passing a sound per urethram and with one finger in the vagina, feeling the point of the instrument in the bladder.

*Causes.* The predisposing cause of prolapse of the vaginal wall is a ruptured perinaeum. The exciting causes are: (i.) increased general intra-abdominal pressure, as in violent muscular efforts; (ii.) a habitually full bladder; or more rarely (iii.) a habitually distended rectum.

*Prognosis.* Prolapse of the vaginal walls in time usually leads to prolapse of the uterus. The *treatment* of the two conditions is very similar (see below).

§ 339. **Prolapse of the uterus** is its displacement downwards. Four degrees of displacement are described—(i.) the organ may occupy a position somewhat lower than normal; (ii.) it may lie wholly in the vagina; (iii.) it may have partly or entirely passed through the vaginal orifice (procidentia); and (iv.) in extreme procidentia it lies entirely outside the vulva, the body lying in the inverted vaginal wall.

In slighter cases the uterus is seen coming down on asking the patient to strain. In severer degrees the os uteri can be seen and the body of the uterus and the ovaries can be felt. The other symptoms of prolapse of the uterus are:—(i.) the uterus is enlarged; the cervix is frequently hypertrophied; there may be accompanying endometritis or endocervicitis; (ii.) there is difficulty in passing water till the prolapsed organ is pushed up; (iii.) sometimes there is a weight or a bearing down feeling in the pelvis, but more often no pain is complained of, and only the discomfort of the lump during walking and sitting is remarked; (iv.) the uterus is usually retroflexed, and therefore the sound is found to pass backwards; (v.) leucorrhœa is usually troublesome. Ulceration of the external parts is apt to supervene on any degree of procidentia.

*Causes.* (1) The predisposing causes of prolapse of the uterus, as in prolapse of the vagina, are:—(i.) a ruptured perineum; (ii.) a relaxed condition of the parts after labour; and (iii.) a laborious occupation which demands much muscular strain, such as that of a washerwoman. The exciting causes are:—(i.) increased intra-abdominal pressure, such as occurs with muscular work and tight lacing; (ii.) the traction from below exerted by a prolapsed vaginal wall; (iii.) the increased weight of the uterus in cases of subinvolution or tumour of the wall.

*Treatment.* Preventive treatment is highly important. Every woman must rest sufficiently long after labour to insure involution of the uterus. All perineal lacerations must be repaired as soon as possible. *Curative treatment.* In slight cases the uterus must be replaced by pushing up first the posterior vaginal wall, then the uterus, then the anterior vaginal wall. Tampons of ichthyol and glycerine are inserted and changed every two or three days. When any inflammation or undue swelling has been reduced by these means and by hot douching, the insertion of a ring pessary is sufficient. In cases where procidentia has occurred a cup and stem pessary may be necessary. After the menopause prolapse may be difficult to cure, because a pessary in the vagina of old people is so apt to cause ulceration. In some conditions surgical interference is called for.

§ 340. **Inversion of the uterus.** Sudden inversion of the uterus may occur in the 3rd stage of labour, when the fundus is relaxed, but here we are concerned only with the chronic form of inversion. Here the fundus alone may be inverted through the os, or the whole uterus may be inverted. (1) The swelling is red, bleeds readily, and is tender. (2) The sound cannot be passed the normal distance, if at all. (3) Bimanually the fundus is found absent; and if a sound is placed in the bladder in the middle line and the finger in the rectum these can be made to meet without any uterus being felt. (4) There may be symptoms of bearing down, menorrhagia, and leucorrhœa. The *Diagnosis* may have to be made from fibroid polypi; but here the fundus is not absent from its usual position.

*Causes.* Chronic inversion of the uterus is an extremely rare condition. It may be (1) the sequel to acute inversion if the patient survive the

shock; or (2) it may be due to the dragging of a fibroid tumour or polypus. This latter may occur with the traction exerted in the operation of removal.

*Prognosis.* There is no tendency to spontaneous cure. Death may occur after a long period of suffering and anæmia, from exhaustion or septicæmia. The *Treatment* is altogether operative, and we must refer the reader to a text-book of gynecology.

§ 341. **Disordered Functions.** Most of the symptoms previously dealt with are in reality disordered functions; but under this heading it is proposed to discuss—(a) DISORDERED MICTURITION (Retention, Frequency, Painful, Difficult M. and Incontinence), (b) PAINFUL DEFÆCATION, (c) PAINFUL SITTING, and (d) DYSPAREUNIA.

a. **Disordered micturition** is dealt with more fully in kidney diseases (§§ 312—314); here only a few of those special to the female will be mentioned.

I. **RETENTION OF THE URINE.** The *causes* peculiar to women are:—fibroids, malignant disease of the cervix involving the vagina, tumours of the vagina, a retroverted uterus (especially when about the 4th month of pregnancy). In these conditions the retention is due to obstruction of the urinary passage consequent on pressure over the mouth of the bladder. The condition is also found in reflex retention after operations on the perinæum, and in hysteria.

II. **FREQUENT MICTURITION** may be produced in women by (i.) pressure on the bladder from a tumour or an enlarged anteposed uterus; (ii.) a vascular caruncle of the urethra; (iii.) cystocele; (iv.) pelvic inflammation, especially during the early stages; (v.) calculi and gravel; and (vi.) various nervous conditions.

III. **PAINFUL MICTURITION** is found especially in connection with urethral caruncle and in the early stages of pelvic inflammation or ovaritis.

IV. **INCONTINENCE OF THE URINE** is found (i.) in vesico-vaginal or vesico-uterine fistula; or (ii.) after dilatation of the urethra has been performed, *e.g.*, as a preliminary to lithotomy.

V. **DIFFICULT MICTURITION** is found (i.) after labour when the parts are swollen and bruised; (ii.) with prolapse of the uterus, in which case the symptom is relieved on pressing upwards the prolapsed parts; (iii.) all causes of incomplete obstruction.

b. **Painful defæcation** may be due to (i.) retroverted and retroflexed uterus, especially when bound down by adhesions; (ii.) an incarcerated retroverted pregnant uterus; (iii.) pelvic inflammation when acute; (iv.) ovaritis; (v.) prolapsed ovary; (vi.) coccydynia; and (vii.) a fibroid or other uterine tumours pressing upon the rectum.

c. **Painful sitting and Coccydynia** is often associated with painful defæcation. (1) The commoner *external* causes of painful sitting are: (i.) a vascular caruncle of the urethra; (ii.) vulvitis and all other acute conditions of the vulva; (iii.) hæmorrhoids or fissures of the anus. (2) The *internal* causes of painful sitting may depend (i.) upon an increased pressure within the pelvis, *e.g.*, pelvic inflammation, or any tumour within the pelvis; (ii.) injury or inflammation affecting the sacro-sciatic and the sacro-coccygeal ligaments; (iii.) a movable condition of the sacro-iliac joints after parturition; or (iv.) a rheumatic condition of the same joints.



(v.) Dislocation, inflammation, or "neuralgia" of the coccyx is also a recognised cause of the condition.

*Diagnosis.* The diagnosis of pelvic inflammation is treated of elsewhere. *Neuralgia* of the coccyx is known by the fact that the coccyx is sensitive to the touch. It may be connected with constipation or disorder of the rectum. Injury of the sacro-sciatic or sacro-coccygeal ligaments is known by (i.) the history of pain often dates from childbirth, or from the injury which produced it; (ii.) pain is produced by pressure on the ligaments, which tightens them; and (iii.) there is an absence of swelling or dislocation of the bone. *Dislocation of the coccyx* has no pain or tenderness, and is known by the fact that the bone, in most conditions, is displaced backwards. When the dislocation is found to be forward, it is much more painful, so that the patient usually sits on one ischial tuberosity—i.e., sits sideways. In a *movable condition* of the joints there is a history of pregnancy with lameness towards the end of gestation, and the patient complains of pain over the pubic bone. In slight cases it may be very difficult to diagnose. *Rheumatism* is known by the absence of other local signs and by the shifting character of the pain, and perhaps the fact that the patient has other manifestations of rheumatism.

*Prognosis and treatment.* Vulvitis and pelvic inflammation are treated of elsewhere. Inflammation and neuralgia of the coccyx are usually cured by laxatives, hot baths, and sedative applications. Injury which has affected the ligaments may also be cured with laxatives and hot baths, but the improvement is slower. Some advise in extreme conditions the division of the ligaments. Dislocation of the coccyx, if backward, may be a cause of no great inconvenience, but if recent may be reduced at the time; if of old standing it should be left alone. A forward dislocation, on the other hand, is much more troublesome, and may require the removal of the coccyx. A movable condition of the joints tends spontaneously to recover. It may be necessary to make the patient rest for a time, and afterwards to walk with a tight bandage across the pelvis.

d. **Dyspareunia** may arise from a variety of causes. (1) Perhaps the most frequent is a functional spasm of the sphincter vaginae, associated perhaps with a general neurotic state. In these circumstances the attempt to pass a speculum will sometimes elicit the same spasm, but may also be a means of cure. (2) Various other local conditions should be carefully looked for, such as a vascular caruncle of the urethra, vulvitis, or vaginitis (see above). Fissures or small ulcers between the folds of the parts, or hidden by the remnants of the hymen, are apt to be perennial causes of discomfort, which will remain undiscovered from month to month, and perhaps year to year. (3) Ovaritis or a prolapsed ovary may produce considerable pain on deep penetration. (4) Parametritis (especially when associated with endocervicitis), perimetritis, and retention of foreign bodies are also apt to become causes of dyspareunia. (5) Masturbation in the female. (6) Finally there may be, though this is relatively rare, a disproportion between the parties concerned.

*Prognosis and treatment.* The condition of dyspareunia is apt to lead to considerable discomfort, not only to the individual, but to home life in general, and may lead to far-reaching consequences; and when at length the aid of the physician is sought it behoves him to make his investigation with the greatest care, and express his opinion with considerable tact.

The first step is to make a very careful and minute examination in a thoroughly good light and under the most favourable circumstances for a local investigation, in view of the minute causes which may underlie the difficulty. The passage of a good-sized speculum will sometimes cure vaginismus. If not, a weak cocaine ointment with small doses of bromide may be tried. Childbirth is a frequent cure of vaginismus and many of the other causes mentioned. The treatment of the other causes will be found elsewhere.

## CHAPTER XV.

### PYREXIA.

#### MICROBIC DISEASES.

WHEN a patient is suffering from some general or constitutional derangement he complains of a vague "feeling of illness" (*i.e.*, malaise), or of "weakness" (debility, asthenia). He feels "generally" ill and perhaps looks ill, but may be unable to mention any localising symptom, such as pain in the side, or palpitation. Now the first thing to do in such circumstances is to ascertain whether he is feverish or not, because all such conditions may be divided into two large clinical groups: A. **Debility with pyrexia**, which includes the Acute Specific Fevers and disorders in which there exists some localised inflammation; and B. **Debility without pyrexia**, which includes the different forms of Anæmia and various toxic and nutritional disorders. The latter will be dealt with in a future chapter; in this chapter we are concerned solely with the various conditions (chiefly microbic in origin) attended by elevation of the body temperature.

§ 342. **Definitions.** The term **Acute Specific Fever** (or Specific Febrile Disease) has been applied to those fevers which are due to a specific or special poison (now known to be microbic) introduced into the body from without, and which run a definite course. If the poison was contracted from a previous case, but without contact with the patient, it was said to be an *Infectious* disease (*e.g.*, scarlatina); if the disease was produced only by actual contact with a person suffering from the malady, it was called *Contagious* (*e.g.*, syphilis); but these terms have always been used somewhat loosely and indifferently. It would be out of place to enter here into the question of the nature of this poison or virus: but suffice it to say that there is direct or inferential proof in all the acute specific fevers that the virus is of microbic origin. At first the microbes themselves were supposed to be the active agents of these diseases; but now in most cases the *causa vera* of the pyrexia and other symptoms is known to be a virus which is given off by the microbe. This branch of knowledge has received literally enormous additions to it during the last quarter of a century (cf. §§ 388 *et seq.*).

The subject of Bacteriology will be referred to in a later chapter, and it will be sufficient here to mention the chief **clinical characteristics**

which cause us to suspect a disease of having a **microbic origin**. They are three in number :—

1. The occurrence of the disease in question in an *epidemic* form, *i.e.*, in the form of an outbreak, or as a series of cases which suggest that they contracted the disease either from one another, or from a common source—the infection being conveyed to them through the air, the water, or other ingesta. Dietetic poisons (organic and inorganic) must be excluded.

2. Two features are common to all microbic diseases :—(i.) *Pyrexia* is present at some time during the course ; and (ii.) all the cases of disease run a *definite course*—acute onset, gradual increase to an acme or fastigium, defervescence gradual or sudden, followed by death or complete restoration to health.

3. The constant presence in the blood, tissues, or excretions of the patient of a *microbe*.

The *pathological proof* that a particular microbe is causally related to the disease consists in applying certain experimental tests (see § 388).

**Epidemic, Endemic, and Sporadic**, are terms by which it is usual to express the relative prevalence of infectious diseases. A disease is said to be *Epidemic* when a large number of cases arise by infection from a common source or from one another at one time, followed by an interval in which none arise. Thus epidemics of measles, scarlatina, and diphtheria arise in the metropolis and elsewhere from time to time. A disease is said to be *Sporadic* when it occurs only in isolated cases. Thus we speak of a sporadic case of Mumps when no other cases of it have been known to occur about the same time and in the same district. An *Endemic* disease is one which is constantly present in a certain district. Thus enteric fever is endemic in London, ague in Central Africa and other marshy areas, and cholera in India.

#### PART A. SYMPTOMATOLOGY.

§ 343. **Symptoms attending pyrexia.** Pyrexia may in some instances be unattended by any symptoms, but in nearly all cases the patient whose temperature is elevated complains of feeling “chilly,” or he may have shivering or rigors ; or perhaps he feels “burning hot.” Headache, restlessness, and vague pains in the limbs and back are also common symptoms, in addition to the malaise or weakness. His skin is hot and dry to the touch, his pulse and respiration are rapid, appetite bad, tongue furred, bowels confined, urine scanty and high coloured. In severe cases of fever there is great prostration, considerable mental dulness, and there may be delirium, or the “typhoid” state. By these various symptoms we suspect the presence of pyrexia, and the suspicion is confirmed, and the degree of fever ascertained, by the clinical thermometer (see below). The various STAGES through which microbic disorders pass, and the three important



symptoms or conditions which are met with in patients suffering from pyrexia—namely, RIGORS, DELIRIUM, and the “TYPHOID STATE”—will now be separately described.

§ 344. **Incubation** and other **stages of microbic diseases.** There is nothing more characteristic of microbic or specific diseases than the *definite course* which they run. The attack of a micro-organism on the human body is somewhat like the invasion of an army which goes on multiplying at a most prodigious rate almost as soon as it lands, and then disappears after it has consumed all the food and burnt up all the available fuel. This simile has indeed been employed, but it is not a happy one, for no army could multiply with such inconceivable rapidity, nor would it be so foolish as to waste 12 days (like Small-pox does) over a period of incubation after it had landed. This is a curious fact not yet fully explained, that a person does not develop the disease directly after he has been exposed to infection. This interval is called the stage of *incubation*. The patient may be quite well during this stage, or feel a little malaise. Its duration is variable in most diseases, and each disease differs from another (Table, p. 574). This period corresponds to the time during which a healthy person who has been exposed to infection needs to be isolated (placed in quarantine as it is called) to see if he will develop the disease. A glance at the first column will show that a period of THREE WEEKS will cover all the eruptive fevers. The actual *invasion* or development of the symptoms of the disease is more or less abrupt, excepting in enteric fever and sometimes measles. An *eruption* appears upon the skin within the next 4 days (excepting in enteric fever), in those diseases which develop a rash, and which are called on that account the Exanthemata. The fever and other symptoms go on increasing until the climax or *acme* is reached. Finally the last stage, the stage of *defervescence*, supervenes, and gradually the patient convalesces.

§ 345. **Rigors** often indicate the onset of pyrexia. A rigor is an attack of shivering attended by elevation of temperature rapidly followed (usually) by sweating and a fall in the temperature. Such an attack may vary widely in severity from a simple feeling of “chilliness down the back, like cold water,” to a shaking of the whole body, so that the patient shakes the bed beneath him. These severe rigors occur typically and *regularly* in the course of

TABLE XXIII.—SHOWING INCUBATION, DATE OF ERUPTION, AND DURATION OF INFECTION OF THE PRINCIPAL INFECTIVE DISORDERS.

DISEASE.	INCUBATION PERIOD.	DAY OF DISEASE ON WHICH RASH APPEARS.	INFECTIOUS PERIOD, or period during which the patient need be isolated.
Varicella	10 to 19 days, average 14	The rash is usually the 1st symptom noticed	Till all scabs have separated. 2 to 4 weeks.
Scarlet Fever	1 to 5 days, average 2½	2nd	From commencement till all peeling ceases. Average 5 to 6 weeks. Otorrhœa may retain infection for <i>six</i> months or more.
Small-Pox	12 days	3rd	From commencement till not a trace left of scabs or desquamation. Most virulent in vesiculation, pustulation, and scabbing. 3 to 8 weeks.
Measles	7 to 14 days, average 10	4th	Great in early period before rash out. Till scaling and cough cease. Average 3 wks.
Rötheln	7 to 21 days, average 10	3rd or 4th, usually 1st	10 to 14 days from commencement.
Typhus	Rarely less than 12	4th or 5th	Probably 3 to 4 weeks.
Enteric	3 to 21 days, average 10	Average 2nd week	Several weeks after pyrexia has ceased.
Dengue	2 to 6 days	Initial rash 1st day Terminal rash 4th	
Diphtheria	2 to 6 days	None	At least 21 days after disappearance of membrane and all throat mischief.

The period of incubation of the other microbic disorders so far as we know is given approximately below. The duration of this period seems to depend on the dose of the poison and many other circumstances, but also on certain conditions belonging to the individual who becomes infected, *i.e.*, of the soil in which the microbe grows. For instance, the tubercle bacillus may be introduced into the lungs, and remain dormant there for an almost indefinite time before producing tuberculosis; and in some cases, where the resisting powers of the individual are sufficient, may never produce it at all.

Ague, 12 hours and upwards.  
 Anthrax, 2 or 3 days.  
 Gonorrhœa, 2 or 3 days.  
 Influenza, 3 or 4 days.  
 Plague, 3—7 days.  
 Glanders, 3—18 days.  
 Relapsing Fever, 4—10 days.  
 Whooping-cough, 6—12 days.  
 Malta Fever, about 9 days.  
 Erysipelas, 3—6 days.

Cholera, under 14 days.  
 Yellow Fever, under 18 days.  
 Tetanus, under 21 days.  
 Mumps, 12—21 days.  
 Syphilis, 15—25 days.  
 Hydrophobia, 40 days or more.  
 Tubercle, probably some weeks.  
 Beri-Beri  
 Pneumonia  
 Septicæmia  
 Cerebro-Spinal Fever  
 Infantile Diarrhœa  
 Psilosis

} unknown.

ague. Severe rigors also occur at frequent but *irregular* intervals throughout the course of Septicæmia. In childhood rigors are often replaced by convulsions.

1. The first thing to do is to ascertain that the shivering is not of nerve origin, because a trembling much resembling a rigor may occur as a result of pure fright or from slighter causes in nervous people.

2. Procure if possible a series of temperature records, because rigors occur in association with several conditions which can only be differentiated in this way.

*Causes.* The causes of rigors are very numerous, but they are best approached in a general way as follows :—

(a) Coming on in a *person previously healthy*, one should always suspect the advent of some acute illness. In children the eruptive fevers are often ushered in with either convulsions or rigors. In adults pneumonia, peritonitis, pyæmia, tonsillitis, the eruptive fevers or influenza may be suspected.

(b) *Septic infection.* When rigors *supervene in the course of an illness* of any kind, abscess or pent-up pus in some position should always be the first thing thought of. *Before the days of the thermometer old authors used to rely upon shivering and sweating as an infallible indication of the formation of pus.* In a case of pleurisy with effusion, for instance, which has hitherto been serous, the occurrence of shivering indicates that the contents of the chest have become purulent (empyema). Similarly, a rigor occurring with otitis media may point to cerebral abscess or sinus thrombosis. Rigors occurring in a case of cardio-valvular disease indicate the formation of emboli, or the supervention of malignant endocarditis (*i.e.*, arterial pyæmia, Wilks). Shiverings and sweatings are apt to occur during the course of tuberculosis and many other conditions mentioned under the causes of Intermittent Pyrexia (§ 378). If no obvious cause for an attack of shivering appears, we may suspect some internal ulceration or suppuration, such as appendicitis, or ulceration in some part of the urinary, biliary, or alimentary canals.

(c) Some *shock to the nervous system* may produce rigors. The passing of a catheter is often followed by a severe rigor, and sometimes the temperature goes suddenly up to 105° or 106° and as suddenly down again. Irritating substances in the alimentary canal may produce rigors reflexly. Sudden obstruction in the biliary or renal passages is often attended by rigors, followed by a

feeling of heat and sweating, and the temperature may go up to  $105^{\circ}$  (Murchison).

(d) *Neurasthenic* and *hysterical* patients are very apt to have shivering attacks, but these are unattended by elevation of temperature. Attacks of shivering may also constitute a symptom of *vasomotor disorder*. It is, for instance, a symptom of the reaction which follows and often forms part of the "flush-storms" chiefly met with at the climacteric—"flushes and shivers," as the patients call them. In these also there is no elevation of temperature.

The *Prognosis* and *Treatment* belong to the several causal conditions, but in any case the patient should be kept warm in bed with a hot water bottle to his feet and a full dose of opium combined with bromide to soothe the nervous system, and in septic or malarial cases a full dose (grs. 5—10) of quinine.

§ 346. **Delirium** is another symptom which frequently accompanies pyrexia. The older authors used to describe 3 varieties of delirium:—(1) *Delirium ferox*, in which the patient is very violent and maniacal. (2) *Typhoid delirium*, in which the patient lies on his back muttering, with *subsultus tendinum*. (3) *Delirium tremens*, in which there is great sleeplessness, hallucinations and tremors, not necessarily due to alcohol. The nature of the delirium is not always constant in any given disease. For clinical purposes, the *causes of delirium* may be divided into two groups—**FEBRILE** and **NON-FEBRILE**. It is important, therefore, to take the temperature at once in every case of delirium. Alcoholic subjects and children are predisposed to delirium when attacked with only slight fever.

a. *Febrile Delirium*, or delirium with elevation of temperature, may arise under 4 circumstances:—

1. **DISEASES OF THE BRAIN**, such as tubercular meningitis. This kind is generally accompanied by pain in the head, vomiting, intolerance of light and paralysis of various cranial nerves.

2. **ACUTE LOCAL INFLAMMATIONS** in other parts of the body, such as pneumonia. It is advisable, therefore, to examine all the organs of the body.

3. All the **ACUTE SPECIFIC FEVERS** are liable to be accompanied by delirium. The tendency, however, varies considerably, and it is important to bear this in mind, because, as a prognostic



indication, delirium occurring in a disease like measles or acute rheumatism, in which it is rare, has a much more serious meaning than when it occurs in small-pox, for instance, where it is usual (see Table XXIV.). Occurring in acute rheumatism it is generally an indication of pericarditis or endocarditis.

4. THE WORST FORMS OF DELIRIUM TREMENS are accompanied by an elevation of temperature. Indeed, the prognosis in this affection very largely turns upon the temperature. We must be careful to exclude local inflammations in such cases, for they are apt to come on very insidiously.

TABLE XXIV.—Showing the relative frequency of DELIRIUM in the various MICROBIC DISORDERS.

Frequent in—	Occasional in—	Rare in—
Confluent Small-pox Typhus Lobar Pneumonia Enteric Fever (after 1st week) Meningitis Cerebro-Spinal Fever Erysipelas Plague Malignant Endocarditis Scarlet Fever Septicæmia	Remittent Fever Yellow Fever Small-pox (modified) Measles Relapsing Fever	Influenza Mumps Dysentery Cholera Acute Rheumatism Ague Diphtheria Rötheln Varicella

b. *Non-febrile Delirium* may arise under 6 conditions:—

1. DELIRIUM TREMENS (Delirium e potu) is, as just mentioned, usually unattended by elevation of temperature, and is therefore the commonest cause of non-febrile delirium. It is recognised by the history, the muscular tremors, sleeplessness, and the marked predominance of hallucinations.

2. CHRONIC RENAL DISEASE, and especially Chronic Interstitial Nephritis, gives rise in its advanced stages to a muttering delirium or incoherence, which thus becomes a symptom of the gravest import, and generally heralds coma and death. The delirium is due to uræmia and occurs in other renal diseases.

3. POST-FEBRILE DELIRIUM (Post-febrile mania). During the convalescence of pneumonia, enteric fever, and other exhausting diseases, especially such as run a protracted course, mental symptoms may develop. These symptoms, which—in most of the cases I have met with—make their appearance without any warning, give great uneasiness to the friends. Nevertheless, by

means of good food, tonics, and fresh air, such mental symptoms will entirely disappear.<sup>1</sup> Before venturing on a prognosis, however, inquiry should always be made for any family history of mental disease, for an hereditary taint greatly lessens the chance of recovery. The condition is recognised by the history of the previous malady. Sometimes the mental derangement consists simply of loss of memory, especially for the names of persons and things, but more often the mind "wanders" and there are delusions.

4. REFLEX DELIRIUM. Trousseau<sup>2</sup> mentions cases of children with intestinal worms who had delirium, and several cases are mentioned by the same author which were caused by the tickling of the soles of the feet. The transient delirium connected with the severe pain of childbirth is probably of the same nature. I am inclined to agree with Griesinger,<sup>3</sup> who says that "mental diseases caused by intestinal worms would be very interesting and more practically useful if they could bear a closer investigation." Nevertheless, the transient delirium or mania met with at the climacteric, comes with some probability in this category; the reflex cause being situated in the generative organs.<sup>4</sup>

5. DELIRIANT DRUGS should always be suspected when delirium develops suddenly in a person in health, especially children in the country, in the absence of any of the foregoing. The most important

<sup>1</sup> The last case of this affection which I have seen, was that of a lady æt. 39, who, after a protracted illness with subacute rheumatism, developed mental symptoms which lasted for some 3 months, until the administration of opium gave her the necessary quiet, and she completely recovered. She had delusions, wanderings at night, and serious loss of memory. She always addressed me as "Dr. Devill."

<sup>2</sup> Clinical Lectures: New Syd. Soc. Translation.

<sup>3</sup> Griesinger on Mental Diseases: New Syd. Soc. Translation, p. 197.

<sup>4</sup> *A case of Transient Mania of the Menopause.* A single lady, æt. 41, of a neurotic temperament, who for 5 or 6 months had been slightly irregular in her periods, which had also been profuse and painful, but who had shown no more definite signs of the menopause than this, remained in bed for some 3 weeks, by my advice, for relief from the pain of a movable kidney, and took a mixture of am. brom. xx grs. and nux. vomica, three times a day. Circumstances compelled her to be a good deal alone. She displayed somewhat amorous proclivities towards her doctor, but was otherwise in her right mind, when, suddenly, during the third week of her confinement to bed, she rushed to the window and commenced shouting incoherently to the crowd who soon collected in the street below. She passed a sleepless night, and was with difficulty kept in bed, talking incessantly; her leading delusions being connected with her own funeral and an imaginary deep well. For six days the incoherence continued, but she gradually became quieter, and got more sleep under the influence of 5 m. tinct. opii every four hours. She was then removed to the quiet of a farmhouse in the country and the society of her friends, where she completely, and to all appearance permanently, recovered her mental faculties in the course of two or three weeks. This seemed to me a good illustration of transient mania connected with the climacteric. No other cause could be made out; unless we are to regard the floating kidney as a source of reflex irritation; or possibly it was the product of the two. There had been no cases of insanity in the family, but her mother had died of "creeping paralysis" at the menopause, and one sister was addicted to alcohol.

are belladonna, hyoseyamus, stramonium and others of the solanaceæ, camphor in rare cases, *ananthe crocata*, *cocculus indicus* (with which beer used to be adulterated), poisonous fungi, and sometimes salicylic acid in large doses.<sup>1</sup> Morphia in some people invariably produces delirium.

6. ACUTE MANIA sometimes comes on very suddenly, and, as previously mentioned, only differs from "delirium ferox" or maniacal delirium in not being referable to some bodily disease or toxic condition of the blood. We are enabled to identify this condition by (1) the temperature not as a rule being elevated; (2) the fact of it coming on in a person previously in good bodily health; and (3) the exclusion of any organic lesion by a careful examination, both of the nervous and other physiological systems. As regards the temperature there is an exception in the rare condition known as "acute delirious mania," in which marked pyrexia is present.

*Prognosis.* As regards *febrile* delirium it is not necessarily a grave symptom when it is associated with a *disease in which its occurrence is usual* (e.g., pneumonia), and especially when the cause is only temporary; but its presence adds considerably to the gravity of a case if the occurrence of delirium is unusual (see Table XXIV.). As regards *non-febrile* delirium, it is a grave symptom in chronic renal disease. The prognosis is serious as regards recovery in all patients who have an hereditary tendency to mental disorder. In acute mania it is relatively bad; but in the other non-febrile conditions above named the prognosis for recovery is favourable.

*Treatment.* It is generally necessary to provide a nurse or attendant, and possibly restraint may be called for. *Remedial treatment.* An ice-bag to the head for an intracranial inflammation; good nourishing food for mania and post-febrile delirium; a brisk purge for uræmia. Alcohol is called for if the pulse is weak, but if it is strong and bounding, alcohol as a rule aggravates the condition. The *symptomatic treatment* consists of sedatives, such as chloral and the bromides. Opium and morphia require caution.

<sup>1</sup> I remember a case of Acute Rheumatism under the care of the late Dr. Murchison when I was his clinical clerk, in a man about 25 years old, who, after taking 20 grs. of salicylate of sodium every 3 hours for only 8 doses, became most violently maniacal, and rushed down the corridor in his night shirt. The drug was stopped and he recovered next day. Careful inquiry was made, but the drug was found to have been absolutely pure.

In delirium tremens, for example, it does a great deal of good in some cases by procuring sleep, but in others it only aggravates the maniacal condition. In post-febrile delirium and other conditions where the brain is suffering from malnutrition, opium in small doses is a most valuable remedy, and may be given without fear if the kidneys are healthy.

§ 347. **The typhoid state** may be described as a condition of unconsciousness (coma) or semi-consciousness attended by elevation of temperature and muttering delirium, due to a toxic condition of the blood. The name of this condition was derived from its frequent association with typhus and typhoid (enteric) fevers. With reference to the question of pyrexia it should be stated that the comatose condition, due to renal disease (uræmia), advanced liver disease, and various poisons (particularly opium), has sometimes been described as the typhoid state, but these are apyrexial conditions and it is preferable to include only pyrexial states under the latter term. In short, the typhoid state corresponds clinically to a state of coma *plus* pyrexia and muttering delirium.

*Symptoms.* The typhoid state is always secondary to some febrile condition, in the course of which it arises. The first symptom usually noticed is sleeplessness with delirium, mostly of the muttering variety, but by-and-bye stupor intervenes, and this gradually deepens. The mental faculties are obscured, but the unconsciousness is not always so complete as one would imagine. The tongue is dry, brown, and rough, and sordes collect upon the teeth. The pulse is rapid, feeble, and irregular, and the heart sounds distant. The respiration is usually rapid, but shallow. The pupils are dilated, but the patient does not see. Nevertheless he looks about at imaginary objects—"coma vigil." Dysphagia may supervene and is a very serious indication of profound stupor. Stertorous respiration only occurs in like circumstances, and is another grave indication. The profound disturbance of the nervous system is evidenced by restlessness, subsultus tendinum (muscular twitchings), floccitatio (picking at the bed-clothes), and, in extreme cases, convulsions. The temperature is elevated, its height and course depending chiefly upon the nature of the primary malady.

*Diagnosis.* (1) The "typhoid state," as above mentioned, may be distinguished from *coma* by the absence of pyrexia, and the



evidences of renal or liver disease, apoplexy, or other cause of the coma. (2) Certain acute *inflammations of the brain* are, however, attended by pyrexia, and offer considerable difficulty. This is particularly the case with tubercular meningitis. The presence of optic neuritis, paralysis of the cranial nerves on the one hand, and the signs of the primary malady which has produced the typhoid condition, on the other, are the only means upon which we can rely.

*Causes.* Patients with an alcoholic history are predisposed to the development of the typhoid state. Renal fibrosis (chronic interstitial nephritis) offers a similar predisposition.

1. The ACUTE INFECTIOUS FEVERS are the commonest causes, and particularly typhoid and typhus. It occurs as an ordinary symptom in the course of these two diseases, and in some others (see Table XXV.). In some other diseases it occurs only occasionally; and in others it is rare. If it arises in either of these latter groups, it indicates either (1) a very severe variety of the disease, or (2) some serious complication; and in any case that the patient is likely to die.

TABLE XXV.—Relative frequency of THE TYPHOID STATE in different diseases. Alcoholic subjects and patients with granular kidney are predisposed to the Typhoid State.

Frequently met with, especially towards the end, in	Occasionally met with in	Rare in
Typhoid (Enteric) Fever	Scarlatina	Measles
Typhus	Diphtheria	Cholera
Confluent Small-pox (unmodified)	Cerebro-Spinal Fever	Variola (modified)
Erysipelas (severe)	Anthrax (Internal)	Varicella
Septicæmia (including Malignant Endocarditis and Osteomyelitis)	Remittent Fever	Dysentery
Meningitis		Ague
Lobar Pneumonia		Relapsing Fever
Acute Miliary Tuberculosis		Acute Rheumatism
Acute Glanders		
Acute Anthrax		
Jungle Fever		
Comatose and Hæmorrhagic Malaria		
Yellow Fever		
Plague		

2. Certain LOCAL INFLAMMATORY DISORDERS may be attended by the typhoid state, such as acute lobar pneumonia, acute pulmonary tuberculosis, ulcerative endocarditis and acute meningitis.

3. Certain acute IDIOPATHIC INFLAMMATORY DISEASES may, in rare instances, be attended by the typhoid state, such as acute

gout and very intense forms of delirium tremens. It is extremely rare in acute rheumatism unless accompanied by peri- or endocarditis.

*Diagnosis of the cause.* The clinical investigation should be conducted on the same lines as in cases of pyrexia—Is it due to *local* or *generalised* inflammation? First, every organ in the body should be thoroughly examined so as to exclude local disorders. Secondly, we proceed to the diagnosis of the general fevers from one another, and, if possible, obtain a series of temperature records. In cases where the cause of the typhoid condition is obscure, septicaemia should always be suspected, and its origin carefully sought.<sup>1</sup> Enteric fever may have to be diagnosed by a process of exclusion.

*Prognosis.* The typhoid state, like delirium, has a less serious import in diseases such as enteric fever, in which it is habitually met with. But it is always a grave condition, and indicates profound cerebral depression. Occurring in the course of scarlatina, erysipelas, or measles, it often indicates pulmonary or cardiac complication, and is proportionately serious. As regards symptoms, the profundity of the stupor is a measure of the intensity of the microbial toxæmia, and dysphagia, stertor or convulsions are generally lethal signs.

*The treatment* of a condition such as this arising in the course of so many diseases must necessarily vary, and our first duty is to *ascertain what disease is in operation*. It is, however, due in all cases to the effects of the poisoned blood upon the central nervous system. The blood poison consists partly of the microbial toxins and partly of the excessive nitrogenous metabolism incidental to pyrexia. The indications are (1) to eliminate the poison by diuretics, diaphoretics, and aperients; and (2) to stimulate and support the patient's strength by nutriment and stimulants. Alcohol was formerly given in large quantities. Dr. Murchison treated patients admitted on alternate days into the London Fever Hospital on opposite methods, and found that they recovered just as well without alcohol; though, on the other hand, it did no

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<sup>1</sup> While I was Medical Superintendent at the Paddington Infirmary a young woman was brought in with all the symptoms of the typhoid state. The subsequent course of the temperature and the occurrence of sweating and rigors declared the disease to be septicaemia, which was traced to a pelvic origin. She died and the case was brought home to a professional abortionist who was sentenced to penal servitude.

harm. In practice, the state of the pulse and of the heart should be our guide. Sinapisms to the feet or calves, strong ammonia to the shaven scalp, cold effusion, are measures that have been employed to arouse the nervous system. As regards symptomatic treatment, if the delirium be very violent, sedatives, such as chloral or bromide, are indicated if the heart will stand them. Opium should be avoided, as it prevents the elimination of the poison. For the treatment of Hyperpyrexia see § 393.

#### PART B. PHYSICAL EXAMINATION.

The clinical investigation of pyrexial disorders consists of—1st, CLINICAL THERMOMETRY; 2ndly, AN EXAMINATION OF THE ORGANS; and 3rdly, BACTERIOLOGICAL INVESTIGATION.

§ 348. **Clinical Thermometry and types of pyrexia.** The temperature is ascertained by means of the clinical thermometer—a little instrument which is now found in most well-ordered households.<sup>1</sup> The temperature of the body is usually taken in the axilla or the mouth. The “index” is first carefully shaken down to a point well below 98·4°; and then the bulb of the instrument is placed under the tongue or in the armpit, and left there for 5 or 10 minutes. The temperature may also be taken in the rectum, where it may be  $\frac{1}{2}^{\circ}$  to 1° higher than in the mouth. The temperature in the mouth is mostly higher than in the axilla, which is best regarded as the normal. In children the thermometer may be held in the groin-fold or “crutch.” The normal temperature of the body varies between about 97·8° and 99°, average 98·4°. It is highest about 8 p.m., and lowest about 4 a.m. It tends to be lower in old age and higher in infancy, especially after an attack of crying. The temperature is subnormal after a loss of blood, during convalescence, in cardiac failure, and in all states of collapse.

A temperature of 100° is regarded as slight fever.

..            .. 102°            ..            .. moderate fever.

..            .. 104°            ..            .. high fever.

..            .. 105° and upwards is regarded as hyperpyrexia.

<sup>1</sup> Owing to the shrinkage of the glass, all glass thermometers are apt after a time to read too high unless they have been stored for months or years before the scale is marked and zero fixed. A clinical thermometer, for instance, may, at the end of a year after manufacture, read a whole degree too high. Hicks, of Hatton Garden, has patented a process of annealing thermometers which obviates this error, and does away with the necessity of prolonged storage. He has also introduced the *facilis index*, the *aseptic thermometer*, and other improvements.

**THE TEMPERATURE CHART.** *Very little information can be derived from a single observation of a patient's temperature, and in all cases of pyrexia one must know the course which it runs from day to day and hour to hour.* In most cases of fever it is hardly possible to come to any conclusion without seeing a "chart" of the case—i.e., a series of records. In all cases of pyrexia the temperature should be taken **and recorded** morning and evening; and in all acute cases it should be taken 4 hourly. In cases of suspected tuberculosis and some other affections it is important to obtain hourly records throughout the day, otherwise slight elevations may be missed. The pulse and respiration should also be observed, especially in abdominal inflammations, where the temperature alone does not give us a true idea of the amount of mischief which is going on. The onset of the pyrexia may be gradual, as in enteric fever or diphtheria, but more often it is sudden and accompanied by a rigor, as in scarlet fever or pneumonia. Remember that the *onset is very sudden* in scarlatina, small-pox, and erysipelas; it is *gradual* (taking perhaps 2 or 3 days) in measles and pertussis. During the next few days the temperature generally increases until the *acme* is reached. The termination may be gradual, when it is said to terminate by *lysis*, as in enteric; or pyrexia may terminate suddenly by *crisis*, as in pneumonia.

**Types of pyrexia.** In the absence of any eruption, the **COURSE OF THE TEMPERATURE** is our best, and may be our only guide. It is usual to describe three types of pyrexia, according to the course which the temperature pursues from day to day (Fig. 93), (i.) *Continued or Continuous Fever*, where the temperature remains at about the same elevation throughout the day, and where the *diurnal variation never exceeds the normal diurnal variation*—viz., one or at most one and a half degrees; (ii.) *Remitting Pyrexia*, when the diurnal variation is greater than the normal diurnal variation, but where the temperature never comes down quite to normal; (iii.) *Intermitting Pyrexia*, where the temperature at some time of the day is normal or subnormal, and at another time of the day, usually in the evening, it is raised 1, 2, or more degrees. But for clinical purposes the two latter may be grouped together, and thus we have **TWO GROUPS** of fevers, one in which the pyrexia is practically **CONTINUOUS**, and another in which there is a remission,



or INTERMISSION, once or oftener during the 24 hours, usually in the morning.

The following are useful facts to remember concerning temperatures: (i.) The sudden advent of high fever in a previously healthy person without other symptoms indicates, in England, Scarlet fever, Small-pox, or Erysipelas, and sometimes Pneumonia. A very gradual advent is suspicious of Enteric fever. (ii.) A fresh rise after the temperature has begun to fall indicates a complication

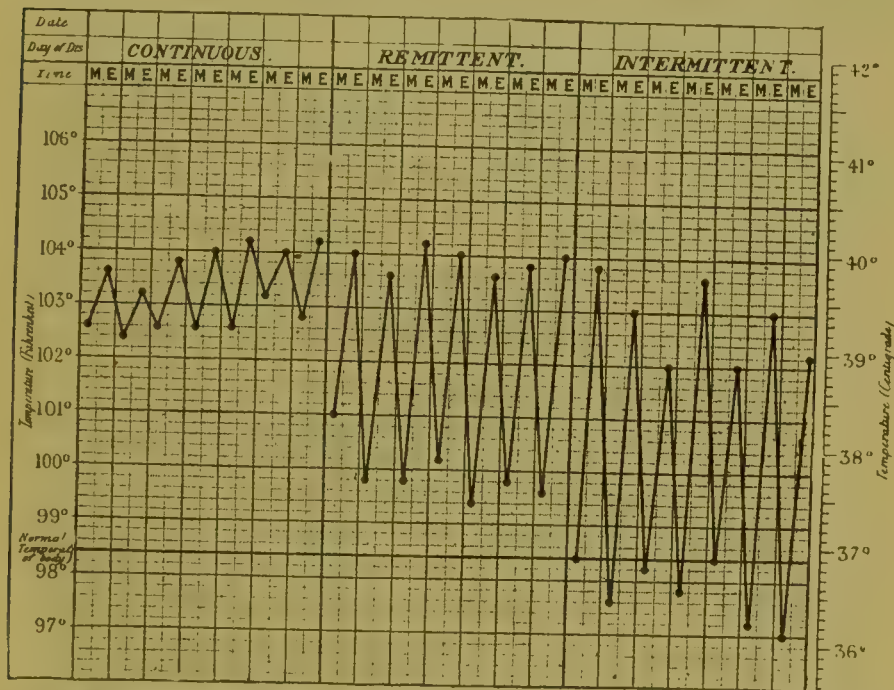


Fig. 92.—TYPES OF PYREXIA. Continuous pyrexia showing only the normal variations in the morning and evening. Remittent pyrexia showing a drop of several degrees each day. Intermittent pyrexia where the temperature comes down to normal at some time every day.

or a relapse. (iii.) A sudden fall in the course of a fever (especially Enteric fever) may indicate internal hæmorrhage, perforation of the peritoneum or pleura, or profuse diarrhœa. (iv.) A considerable rise in diseases usually non-febrile, such as tetanus, delirium tremens, cholera, cancer, epilepsy, apoplexy, etc., generally indicates a fatal termination.

§ 349. **Subnormal temperature.** The temperature of the surface of the body, as indicated in the axilla, is rarely more than one or two degrees below normal. When it is below 96° the condition usually amounts

to collapse. Subnormal temperature is not so important, for purposes of diagnosis, as elevation of temperature: but in the first four instances given below it may aid us in their differentiation. Subnormal temperature adds to the gravity of the prognosis in most wasting disorders. In regard to treatment, temperature readings below the normal are indications for the administration of stimulants and nourishment.

*Causes.* (1) Subnormal temperature as an indication of *lowered vitality* occurs in normal circumstances in the aged, in whom the temperature is habitually several fractions of a degree below normal.

(2) A subnormal temperature is of considerable diagnostic significance in the *prodromal stage of tubercle*, and especially tuberculous meningitis. If a carefully recorded series of temperatures in a person suspected of tubercle show a subnormal morning and a normal evening temperature (or *vice versâ*) it adds to our suspicions.

(3) The temperature takes a *sudden drop* in internal hæmorrhage, or perforation of the bowels. In enteric fever this sudden fall may be the only indication of these serious complications. The rupture of an abdominal cyst, or of an internal organ such as the spleen, liver, or kidney (very rare apart from injury), is attended by a sudden lowering of the temperature; but these conditions are also attended by other and more distinctive signs.

(4) In all severe *abdominal inflammations* prostration and collapse are marked features, and the temperature may in some cases be subnormal, although there may be considerable constitutional disturbance, as shown by the prostration, and the rapid pulse (§ 166).

(5) Subnormal temperature occurs in several other disorders in which it is not of much diagnostic significance, because we depend upon other signs for their identification. Thus, the temperature of the body is lowered—(i.) when there is an excessive withdrawal of heat from the body, as in cases of exposure combined with privation, or with extensive weeping skin eruptions; or when large quantities of fluid are evacuated, as in severe diarrhœa or cholera (when the temperature may be  $90^{\circ}$  in axilla, though  $105^{\circ}$  in rectum); (ii.) in states of inanition or cachexia, *e.g.*, during convalescence from fevers, Addison's disease, cancer (especially of the alimentary canal), diabetes, and chronic mental disorders; (iii.) when there is deficient oxygenation, as in cases of congenital heart disease, cardiac failure, alcoholism, idiopathic anæmia, jaundice, uræmia, pernicious anæmia, and acute yellow atrophy; (iv.) in some diseases of the central nervous system, such as tuberculous meningitis, the onset of cerebral hæmorrhage, or cerebral tumour; and (v.) in poisoning by phosphorus, atropine, morphia, carbolic acid, and other irritants.

(6) In all states of **COLLAPSE** the temperature is considerably lowered ( $2^{\circ}$  or more). Indeed, this is one of the chief means by which it may be distinguished from syncope.

**§ 350. Examination of organs.** All the viscera must be carefully examined in accordance with the scheme of case-taking, pp. 8 and 9, so that local causes for the pyrexia may be excluded. For *clinical* purposes, there are 2 great groups of causes of pyrexia—(a) **local inflammations** such as pleurisy, appendicitis, abscess of the liver, etc., on the one hand, and (b) **general inflammatory**

(constitutional) conditions like scarlatina, rheumatic fever, pyæmia, acute gout, etc., on the other.

In the event of any local inflammation being found, turn to the chapter dealing with the diseases of that part. But it must still be remembered that some constitutional disease (*e.g.*, some fever) *may* be present, of which the local disease is a complication. Thus pneumonia, which would be discovered in the course of our examination, is a frequent complication of enteric fever: and endocarditis of scarlatina or rheumatic fever. There are two features which may lead us to suspect a combination of disorders, such as this—(1) The signs and symptoms of the local disorder may be of an aberrant type (*e.g.*, see *Aberrant Types of Pneumonia*, § 91b); and (2) The constitutional disturbance presented by the patient would be greater in degree or different in kind than would accompany the local disease if it were the only disease present.

§ 351. The examination of blood often affords most valuable information, and it is to be hoped that before very long it will be a routine practice to prepare blood-films in the majority of pyrexial disorders, both for the microscopical detection of parasites and for the investigation of the character and number of the different leucocytes.

a. BLOOD-FILMS. The apparatus is of the simplest—(i.) a sharp double-edged guarded lancet or failing this a sharp needle; (ii.) a spirit-lamp; (iii.) absolute alcohol; (iv.) a glass slide or, if possible, some thin cover-glasses (not exceeding  $18.5\mu$  in thickness) specially prepared so as to be freed from grease—a pair of forceps is convenient for the handling of these latter; (v.) a staining and fixing solution, if the examination is done at the bedside (the most convenient being Leishmann's pulver stain, which fixes and stains at the same time); and (vi.) a  $\frac{1}{12}$ th oil immersion objective. Some prefer to prick the finger behind the nail, but the lobe of the ear is equally convenient and less sensitive. After washing with a little soap and water and then with alcohol, and passing the lancet or needle through the flame, prick the spot and *allow* the blood to flow. It is better not to squeeze the parts as this may disturb the relative proportion of corpuscles. Allow a *minute* drop to touch the centre of the glass slide and immediately spread it out into a very thin film, either with a piece of tissue paper or the edge of another glass slide. If, as I prefer, a cover-glass is used, let the drop of blood touch the centre of one half of a cover-slide and let the half of another cover-glass lie on the top so that the blood spreads out between them—the two clean halves projecting beyond. Then draw them quickly horizontally apart and each will be covered with a thin, equally distributed, film of blood. The film readily dries in the air. Place on it one drop of Leishmann's stain and at once add one drop of distilled water, leave this for 5 minutes, then wash, dry without the aid of heat and mount in the usual way, or examine without mounting under the microscope.

b. To take a SPECIMEN OF BLOOD for bacteriological examination a little U-shaped pipette is desirable, into which the blood will readily run by capillary attraction; when it is full, seal the ends with heat and send to the laboratory.

By a MICROSCOPIC EXAMINATION OF BLOOD-FILMS stained in this way at least two valuable pieces of information can be obtained (i.) the relative number of polynuclear leucocytes (65 per cent.) as compared with the eosinophil cells (about 1 per cent.) and the lymphocytes and other mononuclear cells (taken together 34 per cent.) (Coloured Plate IV.).<sup>1</sup> The percentages given represent the normal proportion of each variety in 100 leucocytes. The relative number of the polynuclear cells is increased up to 80 or more in the presence of any pyogenic process which may be going on. It is very useful to bear this in mind in the diagnosis of a deep-seated or unsuspected pus formation in the presence of an unexplained hectic temperature. (ii.) The presence of any microbes in the blood may be detected at the same time, or the plasmodium of malaria (after a little practice).

**Bacteriological examination** is now almost an essential part of the investigation of fevers; but apart from the blood examination and collection just referred to it involves a highly specialised technique and laboratory apparatus and training.

The PHAGOCYTTIC REACTION of the blood is the relative capacity of the polynuclear white cells for engulfing microbes, and this can be measured by mounting blood-films in the way above described. The estimation of the phagocytic reaction is really a laboratory process, but by a little contrivance it can be done in one's consulting-room if a culture of the suspected microbe is accessible. Take some of the diluted culture, mix it with a drop of the patient's blood on one slide, and take another drop of blood, say, of one's own, which is presumably normal, on another slide, and mix it with another drop of the culture, placing the two slides properly marked in a beaker surrounded by hot water at a temperature of 100°. After leaving them for 10 minutes by the clock, stain and mount both together in the same way (p. 587). By taking a series of microscopic fields and carefully counting the number of microbes each polynuclear cell contains, one can strike an average of the number engulfed, first, by the patient's polynuclear cells and, secondly, by the polynuclear cells in the normal specimen: the ratio between the two is the phagocytic reaction.

PART C. THE DIAGNOSIS, PROGNOSIS, AND TREATMENT  
OF MICROBIC DISORDERS.

§ 352. **Routine Procedure and Classification.** In cases of pyrexia we must investigate, as in other cases, three questions.

*First*, THE LEADING SYMPTOMS complained of by the patient will be some of those mentioned above in § 343.

*Secondly*, THE HISTORY OF THE ILLNESS. The *date* when the symptoms commenced, *i.e.*, the PRECISE DURATION OF THE ILLNESS, is a most important matter. A few of the fevers, *e.g.*, enteric fever and diphtheria, commence insidiously. But the majority are ushered in suddenly, very often with an attack of shivering (a rigor).

<sup>1</sup> See Vol. II.



Throughout the entire course of every case of fever, the physician should have constantly in mind the "day of the disease,"<sup>1</sup> so that he may know what events to expect at that particular period of the case. In enteric fever, for instance, on the 14th day the diurnal range of the temperature should commence to be more marked, and during the next few days special care should be exercised to avoid hæmorrhage or perforation.

*Thirdly*, THE EXAMINATION OF THE PATIENT comprises three important matters—(1) Physical examination; (2) Is there or has there been an eruption? and (3) The temperature and its course.

(1) EVERY ORGAN must be systematically (scheme of Case-taking, pp. 8 and 9), and as carefully and thoroughly examined as the patient's condition will allow, in order that we may DETECT or EXCLUDE ANY LOCAL DISEASE. This is important, because pyrexia may be due to, or manifested by, some LOCAL DISEASE, or some CONSTITUTIONAL DERANGEMENT.

(2) WHETHER THERE IS OR HAS BEEN ANY ERUPTION? is the next question. The first of the groups (*vide infra*) into which all fevers may be divided comprises those in which an eruption distinctive of the disease appears within the first 4 days (with one exception) after the illness. The day on which it appears in each disease should always be at our fingers' ends (Table, p. 573).

(3) THE TEMPERATURE **and its course** is the next thing to investigate; and it is of the greatest importance to obtain a CHART or succession of readings, after the manner described in § 348.

The **classification** of pyrexial disorders may conveniently be based upon the results of our examination, namely, the eruption, if present, and the course of the temperature.

GROUP I.—EXANTHEMATA OR ERUPTIVE FEVERS—*i.e.*, fevers which are characterised by AN ERUPTION distinctive of each disease appearing on one of the first four days of the illness (§ 353).

GROUP II.—CONTINUED FEVERS—*i.e.*, fevers in which the temperature runs a more or less continuous course, and which present NO ERUPTION during the first four days (§ 364).

GROUP III.—INTERMITTENT FEVERS—*i.e.*, fevers in which the

<sup>1</sup> Students do not always understand quite correctly the meaning of this phrase. For instance, the 14th day of a disease is the 3rd day *after* its commencement. Thus the eruption of measles appears on the 4th day, and supposing the patient were taken ill on a Monday, the eruption would appear on Thursday.

temperature runs an intermittent (or remittent) course, and which present NO ERUPTION (§ 378).

If the physical examination reveals signs of disease of some particular organ, reference should be made to § 350, and to the chapter on diseases of that organ.

#### GROUP I. THE EXANTHEMATATA OR ERUPTIVE FEVERS.

In all the diseases in this group the onset of the pyrexia is more or less abrupt, and a well-marked GENERAL ERUPTION appears during the *first four days* of the illness. The course of the pyrexia varies considerably in the disorders in this group. When the eruption comes out it generally drops, excepting in measles and scarlet fever.

##### *Common.*

- I. Chicken pox (1st day) . . . § 353
- II. Scarlet fever (2nd day) . . . § 354
- III. Erysipelas (2nd day) . . . § 355
- IV. Small pox (3rd day) . . . § 356
- V. Measles (4th day) . . . § 358
- VI. Rötheln (3rd or 4th day) § 359

##### *Rare.*

- VII. Dengue (1st day) . . . § 360
- VIII. Typhus (4th or 5th day) § 361
- IX. Anthrax (1st day) . . . § 362
- X. Acute Glanders . . . § 363
- XI. Enteric fever (usually 10th day),  
Influenza, Plague, and other mem-  
bers of Group II., occasionally  
present early rashes.

In each of the exanthemata the ERUPTION has special and DISTINCTIVE CHARACTERS of its own, which, together with the DAY OF THE DISEASE on which the eruption appears, enable one to differentiate the members of this group from one another. SCARLET FEVER may be regarded as the type, but it will be convenient to take them in the order in which the eruption appears. TYPHUS is hardly ever seen in the present day. DENGUE is not met with in England. ANTHRAX and GLANDERS are, like hydrophobia, derived from the lower animals.

§ 353. I. **Varicella or Chicken-Pox** (syn. Variola Crystallina, Spiriae, Nathæ, Ligitime, or Water-pock) may be defined as an acute contagious disease, an eruption of successive crops of large vesicles, usually accompanied by slight exacerbations of fever. It is in most cases a trivial disorder of childhood. A thousand years ago it was described as a spurious form of small-pox (Heberden), and confounded with it till later years.

*Symptoms.* The rash is generally the first thing noticed, though it may have been preceded by a feeling of "chilliness" or feverishness 1 to 3 days before its appearance. It consists of pink, slightly raised papules, which in the course of 12 or 24 hours become vesicular. In the course of a few days, the vesicles enlarge, become depressed in the centre, and finally dry into scabs which fall off in 5 days, rarely leaving any extensive scarring. The essential feature of this eruption is that it comes out in successive crops, and consequently we see different stages of the rash on the same area of skin. This process goes on for about a week, when the disease may be considered to terminate. The rash starts on the chest and neck and

usually invades the whole body excepting the face and hands (the most frequent situations in small-pox). It may invade the mucous membrane. The whole disease seldom lasts longer than 10 days; and it may be so trivial as to pass unnoticed by the patient. The temperature rarely exceeds 103°. A case ceases to be infectious after the scabs have separated. The period of incubation is uncertain, but it is believed to be between 10 and 19 days.

*Diagnosis.* Modified Variola is the chief disease from which it has to be differentiated, although this should not be difficult, because in small-pox (i.) the rash comes out definitely on the 3rd day; (ii.) it does not appear in successive crops; (iii.) its favourite situations are the face and wrists; and (iv.) the constitutional symptoms are very definite and characteristic. *Herpes* is distinguished by the limited area, and grouping of the vesicles. *Pemphigus* is distinguished by the size and chronic character of the blebs. *Dermatitis Herpetiformis* is distinguished by its chronic character, and by the vesicles occurring in groups.

*Etiology.* Varicella is essentially a disease of childhood. It occurs in epidemics; though it is nearly always endemic in London. One attack usually confers immunity, but there are many authenticated cases of second or even third attacks. Other infectious fevers predispose to it.

*Prognosis.* Deaths are very rare. An attack is usually over in a week or 10 days, but it is apt, particularly in adults, to be followed by weakness, which indeed may be more troublesome than the disease itself. *Untoward symptoms*, such as gangrene, and hæmorrhage into the vesicles, are rarely met with. *Complications* are few in number, the chief one being catarrh of the respiratory passages.

*Treatment.* The itching is generally the chief trouble, and this may be relieved by chloral, creolin baths, or calcium chloride. Quinine and arsenic are the best remedies for the resulting weakness.

§ 354. II. **Scarlet Fever** (syn. *Scarlatina*) is one of the most serious, and one of the commonest, of the eruptive fevers. It may be defined as a contagious febrile disease attended by inflammation of the tonsils, and a punctiform eruption on the skin, followed by desquamation. There are 5 characteristic *symptoms*. (1) After a period of incubation which varies from 1 to 5 days, there is a *sudden advent* of high fever. The occurrence of this sudden pyrexia is of itself extremely characteristic of scarlet fever, small-pox, and erysipelas, and, occurring in a child previously healthy, is always suspicious of scarlatina. Vomiting also occurs in the latter in 80 per cent. of the cases (Caiger<sup>1</sup>). The temperature gradually subsides to normal about the 5th day, in mild cases. It does not, as in small-pox, subside when the rash comes out (Fig. 94). (2) A *sore throat* appears on the 1st day, with the fever, and gives a characteristic scarlet colour to the

<sup>1</sup> Dr. F. F. Caiger, in the Article on this subject in Dr. W. H. Allchin's "Manual of Medicine." Macmillan, London, 1900.





by the 7th or 8th day, excepting on the outer side of arms and legs. (4) The *strawberry tongue* is seen typically about the 4th day. It is due to the clearing of the fur, which leaves a bright red surface, with marked fungiform papillæ. (5) *Desquamation* is apt to occur with any severe skin inflammation, but it is more characteristic in this than in any other fever. It begins about the 4th day and continues for 3 weeks—first on the face and, following the order of the rash, *last on the palms and soles*. In the latter positions the flakes are large; elsewhere they are small and branny.

*Varieties.* There are, according to Dr. F. F. Caiger, three chief varieties: (1) The *Benign*, simple or ordinary type as above described. Various symptoms, *e.g.*, rash or sore-throat, may be absent, and these cases are spoken of as *latent*. (2) In *Septic S. F.*, *Scarlatina Ulcerosa*, or *S. Anginosa*, "the ordinary symptoms of *S. F.* are aggravated by the presence of faucial ulceration, which, in addition to being a serious lesion in itself, provides a focus from which septic material is absorbed into the system" (Caiger, *loc. cit.*). (3) The *Toxic* form is a severe variety in which the patient is seized with violent fever, delirium, and perhaps convulsions; the rash is very intense, but the throat symptoms ill-marked, and the patient dies during the first week. *Malignant* or *Typhoid S. F.*, in which there is low muttering delirium, usually a marked rash, and death without complications in a few days, is a very rare variety in the present day. In *Hæmorrhagic S. F.* petechiæ appear under the skin and mucous surfaces.<sup>1</sup> These last are intense varieties of the toxic form.

*Diagnosis.* The diagnosis of scarlatina is not difficult in typical cases. The abrupt advent of high fever, accompanied by vomiting and throat symptoms, in a child who has not had the disease is always extremely suspicious, and if the disease is prevalent the diagnosis is almost certain. During the first few days the greatest difficulty is sometimes experienced in the diagnosis from *quinsy*, in which there is frequently albuminuria at the onset, but less stupor and lethargy, generally less fever,

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<sup>1</sup> *Surgical Scarlatina* is an unfortunate name suggested for an erythematous rash, accompanied by constitutional symptoms, which sometimes occurs in surgical cases. Its nature is uncertain, and many doubt if it be scarlatina.

and the history of previous attacks of quinsy. Slight albuminuria may therefore be present in both conditions during the early stages. Without the eruption it may be impossible to come to a definite conclusion, but in doubtful cases it is best to act as if the graver disease were present (see Table XII., p. 234). *Diphtheria* has no rash, but membrane of a characteristic nature appears on the throat (see Table XII.). *Dengue* (q.v.) is accompanied with severe articular pains and a measly eruption on the 4th day. The diagnosis is easier when the eruption is present. The scarlatinal rash is distinguished from the rare early eruption of *small-pox* by the fact that the latter starts in the groins or axillæ, and is not so bright a red. *Erythema Emetogénés* and *Epidemic Exfoliative Dermatitis* are sometimes mistaken for scarlatina.

*Etiology.* Delicate children, and puerperal cases, have a strong predisposition to the disease. It is a highly infectious malady especially at the outset and during early desquamation. The infection is propagated through the air, and carried by books and clothes; and is not infrequently conveyed by infected milk. The patient is generally regarded as infectious until desquamation has ceased from the palms or soles, a period averaging 4 to 6 weeks. One attack usually gives immunity for a lifetime. The disease is most prevalent during the autumn and early winter.

*Prognosis.* S. F. is always a grave disorder, on account of its possible severity, and the liability to complications, especially renal and ear disease. These dangers are avoided to some extent by keeping the patient in bed. Murchison used to teach that if a patient had been confined to bed 3 weeks nephritis rarely supervened. This point is of the greatest importance, as it is as likely to follow slight as severe cases. After the 4th week there is little danger of nephritis. The aggregate case-mortality under 5 years of age is between 10 and 11 per cent., but it varies in different epidemics. Over 5 it is about 2 per cent.; and is lowest between puberty and 30 years of age. The danger varies with the malignancy of the symptoms, especially the throat symptoms, and the characters of the rash. Persistent vomiting indicates a severe attack. Delirium at night is more or less usual in bad cases, but violent delirium is a bad sign. A temperature of over 105° is a serious symptom. The disease often carries off

the healthy and well nourished, and sometimes spares the delicate patient; but in the *puerperal state* and in those with *tubercular* tendencies, especially the former, the prognosis is very grave.

The *Complications* and *Sequelæ* are extremely important, for they may cause death even after slight attacks. A considerable change has taken place in the nature of the complications and sequelæ met with in modern years, chiefly owing to improved methods of treatment. Acute nephritis and tonsillar abscess used to be regarded as the chief dangers, but at the present day Dr. Caiger<sup>1</sup> gives otorrhœa and otitis media as the chief *complications*, attacking nearly 15 per cent. of all cases, and leading in many to defects in hearing, intracranial abscess, septic thrombosis, and other septic conditions. Next in order come simple albuminuria, attacking 8.26 per cent., definite acute nephritis only 3.12 per cent., and cervical adenitis 5.66 per cent. Acute nephritis appears usually at the end of the 3rd week, very rarely after the 4th, its advent being indicated usually by vomiting and dropsy. Various forms of arthritis (3.75 per cent.) and secondary tonsillitis (2.36 per cent.) are met with chiefly among adults. The other proportions given by Dr. Caiger are—various forms of abscess, 2.58 per cent., ulcerative stomatitis, 2.11 per cent., broncho-pneumonia and bronchitis together, 2.54 per cent. Brawny swelling in the neck, cancrum oris, and noma pudendi are occasionally met with, and may need prompt surgical measures. Acute endocarditis and septicæmia never occur among the cases treated at the Metropolitan Asylums Board's Hospitals, but they are still liable to complicate cases treated in their own homes. Among the *sequelæ* acute and subacute rheumatism (or a septic arthritis very closely resembling it) and chorea are perhaps the chief. Post-febrile mania is also an occasional sequela.

*Treatment.* The general treatment is dealt with in §§ 392 *et seq.*, but it will be well to make a few remarks on the symptomatic treatment. The throat is best treated by a chlorine gargle (F. 18). In children this may be applied by means of a syringe or a spray; nitrate of silver and other caustic applications so long in vogue only aggravate the condition. For the glandular swellings apply glycerine and belladonna, or warm fomentations. Brawny

<sup>1</sup> Dr. F. F. Caiger. The complications of scarlet fever based upon an examination of 30,417 consecutive cases treated in the Hospitals of the Metropolitan Asylums Board. Allchin's "Manual of Medicine," vol. I., p. 286.

swelling of the neck is a serious complication, and must be dealt with by early incision and frequent carbolic fomentations. For convulsions give purgatives (croton oil) and a hot air or steam bath, or a hot water bath. The patient should be kept in bed for three weeks, whether the attack be slight or severe, chiefly to prevent renal complications. As regards *immunisation*, the microbe of Sc. F. has not been isolated, so that we are not yet in possession of an antitoxin.<sup>1</sup> Nevertheless, in view of the numerous septic complications of the disease, antistreptococcus serum has been tried.<sup>2</sup>

The *hygienic* treatment is considered in §§ 390 *et seq.*, but a study of the long list of infective complications given above will show how important it is to treat this malady in a large and airy hospital instead of at home. The throat is in a highly vulnerable condition, and it is doubtless through this portal that the various infective organisms find entrance.

§ 355. III. **Erysipelas** (Synon. The "Rose," or "St. Anthony's Fire") may be defined as an acute febrile contagious disease, characterised by a progressive margined redness and tumefaction of the skin, usually attacking the face, or the neighbourhood of a wound). (1) *The stage of invasion.* After an incubation period of 3 to 6 days, the advent is abrupt, as in small-pox and scarlatina. The temperature on the evening of the same day may be 103°—104° or more. Vomiting at the advent is very common, and so also are muscular pains, especially pain in the back,<sup>3</sup> like that of small-pox. (2) *The eruption* begins about 24 to 36 hours after the advent of fever, as a red spot on the face or at the site of an abrasion (which may be microscopic). It enlarges, spreads, becomes bright red, tender, and pits on pressure. The advancing edge is sharply defined and raised, the receding edge indefinite. The eruption may vary in duration from 3 or 4 days to a fortnight. Delirium at night is not unusual. Convalescence becomes established and desquamation occurs in the course of one to three

<sup>1</sup> Dr. W. J. Class has isolated a diplococcus having specific characters from the throat and blood of S. F. patients: *The Lancet*, Sept. 29, 1900. Gordon and Klein have isolated a streptococcus.

<sup>2</sup> See a case of S. F. complicated by acute Otitis Media treated by Anti-Streptococcus serum, with recovery: Low, *Lancet*, 1898, vol. i., p. 779.

<sup>3</sup> This is not usually mentioned as characteristic of Erysipelas, and the first case I was called to I mistook for small-pox on this account. I have never met with a case in which it was absent, excepting in 2nd or 3rd attacks of the disease.



weeks. During this last stage albumen may appear in the urine, if it has not appeared before.

*Diagnosis.* Erysipelas is to be diagnosed from *erythema* complicated by cellulitis, in which the margin is less raised, and there is less fever. *Zoster* of the first division of the 5th nerve has vesicles in groups, is limited to one side of the face, and unattended by fever.

*Varieties.* (i.) Phlegmonous erysipelas or gangrenous erysipelas are severe varieties with suppuration or extensive sloughing. (ii.) Erysipelas neonatorum is a very fatal variety; death may be due to peritonitis by inflammation spreading along the umbilical cord; (iii.) Erysipelas of the fauces is a severe variety, the eruption spreading to, or starting in this situation. The disease may spread to the larynx and cause fatal dyspnoea.

*Etiology.* It is a highly contagious malady, and spreads through the air to a considerable distance. Persons are predisposed to it by wounds and unhygienic conditions. It seems possible that even in so-called idiopathic cases the virus is introduced into the system through a minute and hardly visible scratch. The presence of a wound is the strongest predisposing cause, and it spreads amongst surgical patients with great rapidity. As regards age, infants and persons over 40 are most liable. A streptococcus (coloured plate at end of work) is the cause of the disease, but its identification with, or relation to, streptococcus pyogenes is undecided. One attack gives no immunity; on the contrary, it predisposes, and some elderly people are liable to an attack of facial erysipelas every year.

*Prognosis.* The usual course is favourable, but the disease is dangerous in infancy or old persons, alcoholic or plethoric patients, and those affected with chronic diseases. Death may occur by coma or syncope, preceded by incessant vomiting; or by the supervention of complications. Hyperpyrexia, persistent vomiting, lividity of the rash, and typhoid delirium are untoward symptoms.

*Complications.* (i.) Subcutaneous abscesses either on the scalp or in the neck; (ii.) diffuse cellulitis, ending often in extensive sloughing; (iii.) acute œdema of the glottis from the extension of the eruption (a very serious complication); (iv.) hypostatic congestion of the lungs (very common), bronchitis, lobular pneumonia,

pleurisy; (v.) peritonitis, especially when it occurs after parturition, and gastro-enteritis; and (vi.) nephritis, acute or chronic, though it is not so common as after S. F.,—are some of the commoner complications. (vii.) Meningitis used to be mentioned as a frequent complication on account of the frequency of cerebral symptoms in Erysipelas; but meningitis does occasionally occur. (viii.) Chronic ulceration or skin eruptions often disappear after an attack of erysipelas near them. This has happened even in the case of ulcers which have been of a malignant character.<sup>1</sup> (ix.) Pyæmia and ulcer of the cornea are among the sequelæ.

*Treatment* (Hygienic treatment, see §§ 388 *et seq.*). A mild aperient should be given when the eruption comes out, and this should be followed by iron in large and frequent doses—20 minims of the liquor every 4 hours. Ammonia and bark are sometimes given; and in Germany large doses of quinine. Alcohol may be required in large quantities. Warburg's tincture is useful. Tannin or liq. ferri. perchlor. are applied locally for the pharyngitis.

*Local treatment.* Antiseptics, or a dusting powder of starch and zinc oxide, or a lotion of acetate of lead and extract of opium (gr. iv. of each to the oz.), should be applied to the inflamed area. The eruption may sometimes be stopped by a subcutaneous injection of carbolic lotion 1 in 20 along the margin. Some say it may be stopped by painting the advancing edge with nitrate of silver. Daily inspection must be made for abscess whenever the skin is tense; scarification relieves the tension, and may prevent the occurrence of suppuration. *Immunisation* is now obtainable by the use of antistreptococcus serum, and cases have been cured in this way (§ 388).<sup>2</sup>

§ 356. IV. **Small-pox** (*Variola*) is a highly contagious eruptive fever, the eruption passing through the stages of papule, vesicle, pustule, and scab. In small-pox UNMODIFIED BY VACCINATION the symptoms are as follows: (1) After a very definite period of incubation of 12 days, characteristic constitutional symptoms occur, viz., sudden advent of high fever (101°—104°), with severe headache and *pain in the back*. The most noticeable features of this primary fever are the severity of the pain in the back (which, in

<sup>1</sup> It has been suggested to inoculate erysipelas as a means of cure in this condition (*vide* Carcinoma).

<sup>2</sup> Washbourn: *Lancet*, 1899, vol. ii., p. 1010.





*Small Pox.*

Mab. Green, *del.*

Right side of face (l. of observer) represents the 2nd day of the eruption; the other side represents the 6th day of the eruption. A few of the pustules show commencing umbellication.  
*The picture should be viewed at a little distance.*



my experience<sup>1</sup> is present even in the mildest cases) and the frequent occurrence of vomiting.<sup>2</sup> The fever remains up until the 3rd day, when the eruption appears; it then drops considerably, the patient indeed may feel comparatively well. About the 7th or 8th day, when the spots become pustular, a secondary or suppurative fever develops, which may be attended by rigors (Fig. 95). This secondary fever lasts 6 or 8 days. (2) The eruption appears between the 3rd and 4th day after the illness has commenced (14 days after infection), first as a crop of

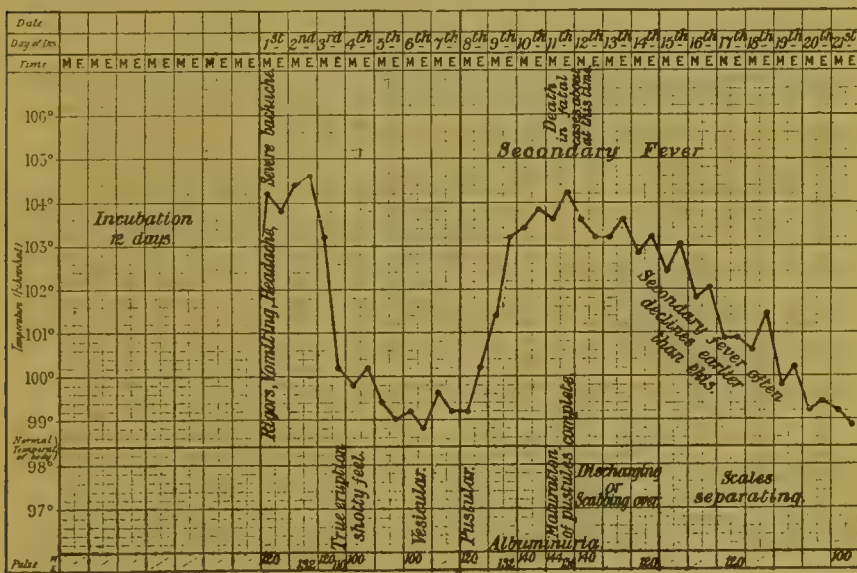


Fig. 95.—UNMODIFIED SMALL-POX. Severe confluent case, unvaccinated, terminating in recovery. The various incidents are shown in the chart, for which the author is indebted to Dr. F. F. Caiger.

papules of *shotty hardness*, which can be felt even more readily than they can be seen, like small shots beneath the skin (Coloured Plate I.). They first appear on the face, and then the eruption travels downwards over the whole body. Two days later the papules become vesicular. The eruption comes out in one crop, and is therefore never multiform in any given area of

<sup>1</sup> Report on the Warrington Small-pox Epidemic, by Dr. T. D. Savill; Blue Book of the Royal Commission on Vaccination. Eyre & Spottiswoode, London, 1893.

<sup>2</sup> During this stage of primary fever there is, as a rule, no eruption, but in a few cases an initial rash makes its appearance. This may be (i.) erythematous, generally found in the groins or other folds, occasionally it covers the whole body; or (ii.) a hemorrhagic eruption sometimes appears on the anterior surface of the abdomen and thighs. An initial rash appears about the 2nd day.

skin, as it is in varicella. Each vesicle enlarges, and by the 6th or 7th day has become pustular, presenting in typical cases, unmodified by vaccination, a depressed centre which is held down by a bridle in the middle, a feature known as umbilication. The next day (8th day) the bridle ruptures, and each pustule becomes hemispherical, about as large as a split pea, with an inflamed and indurated base. These pustules gradually dry into

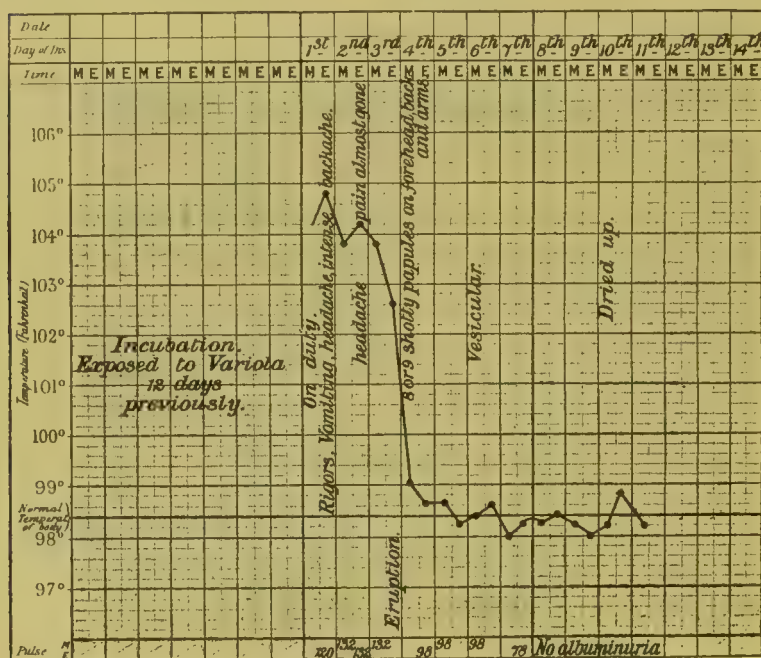


Fig. 96.—A mild case of MODIFIED VARIOLA occurring in a young woman, *æt.* 22, who had been vaccinated two years previously and who presented three visible cicatrices of the primary vaccination. Initial symptoms severe. **No secondary fever.** The author is indebted for this chart to Dr. F. F. Caiger.

scabs, which fall off about the 15th to the 20th day, leaving patches of congested skin, and in severe cases a pitted cicatrix. The extent of the eruption and the amount of inflammatory induration varies considerably. Sometimes only the face and wrists present a few spots; sometimes the whole body is covered. The eruption on the legs always presents a proportionate retardation of development, since it appears last in this situation. Consequently, before certifying a patient as free from infection, the

soles of the feet should be carefully examined, and found free from desquamation.

MODIFIED SMALL-POX, or VARIOLOID (Fig. 96), is the term applied to the disease when modified by previous vaccination. The primary fever and early symptoms are indistinguishable from the unmodified form above described, and the eruption appears on the third day. Modified differs from unmodified small-pox in four ways—(i.) There is no secondary (suppurative) fever; (ii.) certain portions of the eruption abort and do not pass through all stages; (iii.) as a consequence, several stages of the eruption may occasionally be seen on the same portion of skin; and (iv.) the general eruption may be very scanty, and may consist of not more than a dozen papules.

*Varieties.* It is sufficient to make 3 varieties, according to the severity of the disease, the severity of the symptoms corresponding very closely with the character and extent of the eruption: (1) *Mild*, (2) *Discrete* and (3) *Confluent*. The *Hæmorrhagic* and *Corymbose* varieties are very severe and, fortunately, rare; these adjectives referring to the character of the spots. In *Malignant* S. P. there are hæmorrhages into and beneath the skin and death ensues early (Fig. 97).

*Diagnosis.* In modern times, when nearly all cases of S. P. are modified by vaccination, the diagnosis is not always easy, and the greatest difficulty may be experienced in making a diagnosis even from Acne. There are 3 important diagnostic features: (i.) sudden advent of high fever; (ii.) headache, *backache*, and vomiting at onset of the disease, of which there should always be a history, even in the mildest cases; and (iii.) the shotty character of the papules.<sup>1</sup> *Measles* is the disease which is most often mistaken for S. P. in the early stages of the case, and therefore 2 plates of these diseases are presented side by side (Coloured Plates I. and II.). *Measles* is distinguished from S. P. by the redness of and the running from the eyes, and by the fact that in 24 hours the papules become smaller, whereas in S. P. they become larger. *Varicella* is distinguished from S. P. by the inappreciable character of the premonitory constitutional symptoms; by the eruption coming out in a succession of crops, so that one portion of skin may show several stages of the eruption; and by the vesicles not being preceded or accompanied by any *shotty induration*. In febrile *roseola* or lichen, the fever lasts only 24 hours, the efflorescence

<sup>1</sup> See also a paper by the author "On the Diagnosis of the Early Stage of Small-pox," *Brit. Med. Journ.*, April 29th, 1897.

appears all over the body at once, and it does not go on to any further stage. *Pustular syphilide* is chronic, and is unattended by any marked pyrexia.

*Etiology.* The malady is highly infectious, but the microbe has not yet been discovered. Children, and especially infants, are particularly prone to the disease, and before the discovery of

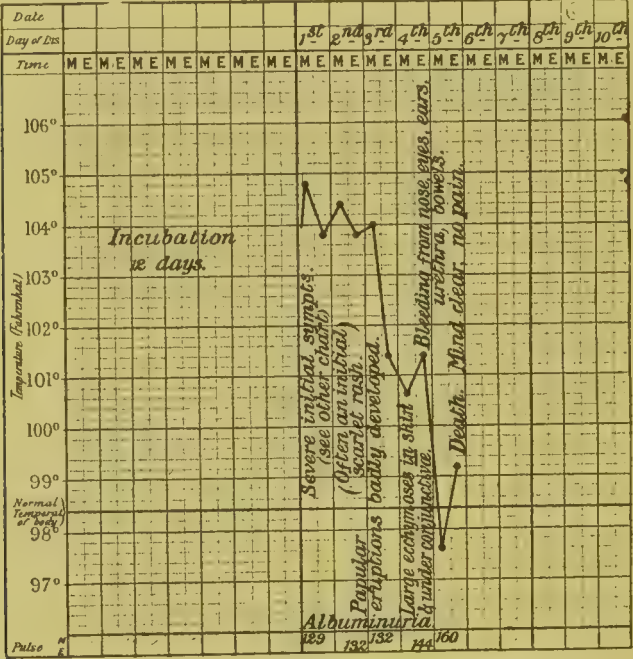


Fig. 97.—A case of MALIGNANT HEMORRHAGIC SMALL-POX (as distinct from those cases of confluent small-pox with hemorrhages in the pustules). Patient unvaccinated. Death occurred on the 5th day. The various incidents are shown on the chart, for which the author is indebted to Dr. F. F. Caiger.

vaccination (A.D. 1776) it was a cause of considerably more than half the infantile mortality in Great Britain and other countries.<sup>1</sup> The poison is conveyed through the air to a considerable distance. Some believe it may be conveyed to a distance of miles, but this

<sup>1</sup> It is a fact of some interest that Warrington was the scene of an epidemic of Small-pox in 1773, and the death-rate from the disease in that year was 26.5 per 1,000 (211 deaths, and reckoning 5 inhabitants to a house, 8,000 inhabitants), all the deaths occurring in persons under 9 years of age. (Dr. Thomas Percival, F.R.S., Phil. Trans., 1774, vol. lxxv.) In 1892-1893 Warrington was again visited by an epidemic of S. P., and the death-rate was then 1.1 per 1,000 of the inhabitants, who at that time had only about 1 per cent. unvaccinated persons among them.



is very doubtful.<sup>1</sup> One attack confers immunity; authenticated second attacks of S. P. are extremely rare.

*Prognosis. Vaccination.*—The case-mortality of S. P. in the present day is about 37 per cent. amongst the *unvaccinated*; about 5 or 6 per cent. amongst all classes of the *vaccinated* taken together; and about  $\frac{1}{2}$  per cent. amongst the *properly vaccinated*. The severity of the disease seems to depend almost entirely upon whether the patient has been recently and efficiently vaccinated.<sup>2</sup> In the healthy and recently vaccinated it is a comparatively trivial disorder, but in the unvaccinated, especially in infancy, it is one of the gravest diseases. The second factor in the prognosis is the question of *age*; and the official records of the unmodified outbreak in Warrington in 1773 show that of 211 fatal cases 166 were under 3 years of age. Alcoholism, plethora, and scrofula add to the gravity of the disease. The greatest danger is on the eighth day in Discrete S. P., and on the eleventh day in Confluent S. P. As regards the *varieties*, Confluent S. P., in which the rash comes out on the second day and is very abundant, is much more dangerous than the Discrete form. In the former, the fever does not subside on the third day, and there is a great tendency to hyperpyrexia and complications. As regards untoward *symptoms*, the more severe the primary fever in the unvaccinated, the more severe will be the disease, but this is not so in the vaccinated; profuse salivation is a bad symptom; the case is grave if there be no swelling of the skin, and still graver if the swelling goes suddenly away; convulsions and other complications are unfavourable.

*Complications.* (i.) Acute laryngitis or œdema glottidis is a common cause of death. Hypostatic congestion, pleurisy, empyema,

<sup>1</sup> This question has been very hotly debated, but in the author's belief there are no definite evidences of S. P. being conveyed through the air to a greater distance than a few yards. It is extremely contagious, and all the cases supposed to be due to aerial spread can, if sufficient information can be procured, be explained by the conveyance of contagion either from person to person or through some mediate agency. (Report on the Warrington Small-pox Epidemic, 1892—1893, pp. 64 to 77. Appendix to the Report of the Roy. Com. on Vaccination.)

<sup>2</sup> The figures from the Warrington epidemic 1892—1893 are very striking. In the *infected* houses there were 2,535 persons, and 2,223 of these persons had been vaccinated in infancy. Among these latter 521 (23·4 per cent.) were attacked, and 27 died, so that the case-mortality among them was 5·2 per cent. There were in the *infected* houses 107 unvaccinated persons of whom 60 (56·1 per cent.) were attacked, and 21 died, giving a case-mortality of 35·0 per cent. The figures also showed that in proportion as the vaccination had been more efficient the severity of the disease was less. Finally, among all the 667 cases which occurred in this epidemic not one had been vaccinated or re-vaccinated within 7 years of the attack. (Appendix to the Report of the Roy. Com. on Vaccination, 1894.)

and pneumonia are apt to occur. (ii.) The heart may be affected with peri- or endo-carditis ; but myocarditis and granular degeneration are more common ; (iii.) ophthalmia and consequent destruction of the eye is common in the East—painless corneal ulcers may form and perforate ; and (iv.), for the rest, the complications are the same as those of scarlet fever, but nephritis is not so common.

*Treatment.* It should be remembered that vaccination is capable of modifying the disease even after exposure to infection, because the incubation period of S. P. is 12 days and that of Vaccinia only 8 days. Vaccination may, therefore, be performed with efficacy during the first 3 or 4 days after exposure ; and every member of an infected household should be vaccinated immediately S. P. breaks out therein. As regards *therapeutic agents* little is necessary, in the Discrete form, beyond a mild aperient and salines. In the Confluent form stimulants are necessary, and we must watch for complications and meet them as they arise. If much salivation be present, it may lead to suffocation ; the patient should be put into a warm bath and kept there for a considerable time. For sore-throat use gargles ; for œdema glottidis, inhalations, or tracheotomy may be necessary. Many devices have been contrived to *prevent scarring* by the eruptions, such as powdering with zinc and starch powder, or with pulv. cretæ aromat., with a small quantity of disinfectant, or laying on lint soaked in glycerine and water, with a drop or two of carbolic acid. But all of these are of very doubtful benefit. There is, however, a method which promises to be really efficacious, namely, placing the patient in a room from which all but the red rays of the spectrum are excluded by pasting red paper over the windows. *Hygienic treatment* is given in §§ 390 *et seq.*

The *Preventive Treatment* of Small-pox is accomplished in the present day by three means—disinfection, isolation, and vaccination. Concerning the first two see § 390, the efficacy of vaccination in the prevention and modification of S. P. see p. 603 and below. The *inoculation of S. P.* used to be practised because it was found that the inoculated disease was milder, and gave just as much immunity from a second attack. Out of 20,000 inoculated by the brothers Sutton not one died. It was, however, declared illegal in 1840.

§ 357. **Vaccinia.** VACCINATION is the production in a person of the disease called vaccinia, by inoculating him with the lymph taken from the udder of a cow or calf suffering from that disease. It was noticed in 1769 by a German that people engaged in the milking of cows were exempt from S. P. Jenner in 1775 and 1776 reduced the subject to scientific principles, and ascertained that the inoculation of a human being with the lymph taken from the unbroken vesicles on the udder of a calf suffering from vaccinia protected that person from small-pox. He was also the first to inoculate this disease (vaccinia) from person to person by taking the lymph from the vesicle on the arm which had matured on the 8th day after inoculation. Vaccination was made compulsory in 1853. In 1897 this law was repealed in response to an outcry among the public that syphilis and (?) other diseases could be conveyed from person to person in this way. Syphilis certainly has, in rare instances, been conveyed by arm to arm vaccination ; but by using calf lymph this is entirely obviated ; and all public vaccinators now use lymph direct from the calf. Anyone who now goes before a magistrate and solemnly declares that he has "conscientious objections" to vaccination can procure exemption for himself and his children from compulsory vaccination.

*Rules for vaccination.* Calf lymph is now universally used.<sup>1</sup> The best method is that of scraping the cuticle with a blunt pointed lancet ; the lancet should be kept scrupulously clean, and passed through a flame before using. The doctor's hands should be clean, and the arm of the patient should be washed with soap and water before vaccination.

*The phenomena of vaccination, i.e.,* the symptoms of the disease vaccinia or cow-pox are *nil* for the first 2 days. On the second or third day a slight pimple, on the fifth day a bluish-white cupped vesicle appears, and on the eighth day (the same day of the week in which the operation was performed) the vesicle *becomes matured*. It should never become purulent, but the areola increases during the next 2 days. The contents then become

<sup>1</sup> If human lymph is employed it should be taken from a child, not an adult, and the child should be in good health, and free from any evidences or history of syphilis. The lymph should be taken from a vesicle before it becomes opaque, and before the areola has formed. It is better to vaccinate from arm to arm than from stored tubes.

cloudy, and after the tenth day they dry up ; the scab falls on the fourteenth or fifteenth day, leaving a pitted cicatrix.

The inquiries which the author made on behalf of the Royal Commission on Vaccination into the Warrington Epidemic (*loc. cit.*) went to prove—(1) that primary vaccination offers absolute protection against *infection* for the ensuing 5 or 6 years, and relative protection (gradually diminishing) for a considerable time ; (2) that primary vaccination lessens the *severity of the attack* of S. P. if contracted during the ensuing 20 or 30 years ; (3) that re-vaccination affords absolute *immunity from attack* during the ensuing 5 or 6 years and relative protection for the rest of life ; and (4) that if everybody were vaccinated in infancy and again at 12 and 21 S. P. would be exterminated.

§ 358. V. **Measles** may be defined as an infectious febrile disease attended by catarrh of the respiratory passages, and by an eruption of minute elevated papules aggregated into irregular and often crescentic groups.

*Symptoms.* (1) After an incubation period of 7—14 days, the pyrexia (Fig. 98) comes on abruptly, though not so suddenly as in scarlet fever, rising to  $102^{\circ}$  or  $103^{\circ}$  on the evening of the first day. The next day it usually declines a little. When the rash appears on the 4th day it rises again, remains up until the 6th day, and then falls by crisis. (2) The fever is attended by symptoms of coryza—for which indeed the case may be mistaken if the temperature be not very high. There are profuse lachrymation, running of the nose, and bronchial catarrh, the larynx and bronchi being specially involved. The fauces are sore, and mottled with redness, but not much swollen.<sup>1</sup> (3) The *eruption* appears on the 3rd or 4th day (Coloured Plate II.). It consists of red, raised, well-defined flat papules, discrete at first, but afterwards running in patches. The colour is a reddish-brown, disappearing on pressure. The spots first appear on the *face* and side of the neck, where they are most abundant, and then pass downwards. Each papule reaches its maximum in about 12 hours, and then feels soft and velvety, thus differing from the

<sup>1</sup> The eruption has been said to appear upon the buccal mucous membrane before it appears upon the skin by 1, 2, or 3 days ; and this may be a valuable aid to diagnosis—Filatow, Koplik, and Falkener (Medical Supplement to the Metropolitan Asylums Board Report, 1899).





Mab. Green, del.

*Measles.*

The eruption is 12 hours old (2nd day of rash); the rash represented on the left temple is 24 hours



early stage of small-pox papules. In 12 hours' time they begin to recede, and at the end of 48 hours to fade. By the 8th or 9th day the eruption of measles has completely disappeared, excepting that a brownish mottling of the skin may remain for some time after. Occasionally the macules become petechial. Sometimes the eruption suddenly disappears—the result of some internal complication, not, as is often supposed, the cause. The catarrh goes on increasing during the development of the rash, and

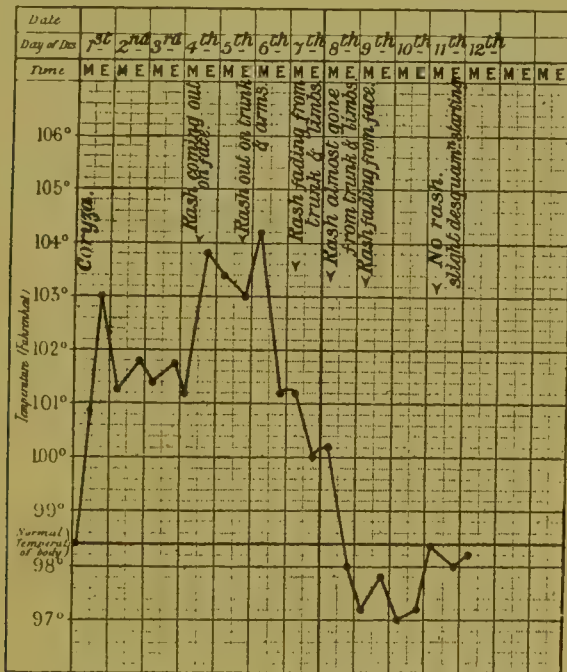


Fig. 98.—MEASLES. Ethel H—, at. 5 (under the author's care). Typical chart. The various incidents are shown upon the chart.

they subside together about the 7th or 8th day, when convalescence commences. Slight desquamation of minute flakes, chiefly on the face, occurs occasionally.

The *varieties* are less well defined than in Scarlatina. The malignant or hæmorrhagic variety is a very severe form, and is attended by petechiæ and the typhoid state. The rash and catarrh may be absent respectively in rare cases.

The *diagnosis* from a severe “catarrh” is very difficult until the eruption appears. *Variola* often presents a difficulty, though the

absence of catarrh, and the presence of pain in the back and vomiting, aid us considerably in diagnosing variola (see Plates I. and II.). The differences between the rashes are referred to above. *Urticaria* is somewhat like measles, but is recognised by the absence of catarrh and pyrexia. For the diagnosis of German Measles see § 359.

*Etiology.* Measles is essentially a disease of childhood, and few escape. It is endemic in England, and outbreaks occur from time to time. The seasonal prevalence is in the spring and winter. The essential cause is a specific microbe, which has not yet been satisfactorily identified. It is conveyed chiefly by the breath and nasal mucus. Unlike scarlatina, it is as contagious before as after the eruption has appeared. One attack confers relative immunity; second attacks are less common than in scarlatina. The majority of so-called second attacks are probably Rôtheln.

*Prognosis.* Measles is not a serious disease in itself, excepting in infancy. The case-mortality does not often exceed 1 or 2 per cent., though it may be as high as 10 or 12. The chief danger of the disease rests in the complications and sequelæ which may attend even the mildest case of measles. The prognosis is bad in proportion to the severity of the pyrexia and pulmonary symptoms. Strumous or weak children suffer most. Convulsions late in the disease are of grave significance. The most important and most common *complications* are bronchitis, broncho-pneumonia, pneumonia, and collapse of the lung. Phthisis is a recognised sequela; it follows measles and whooping-cough more frequently than any other febrile disease. Catarrhal laryngitis, diphtheria, and laryngismus also occur. In all cases of measles with sudden aggravation of fever and no apparent cause, the presence of acute otitis media may be suspected. Cancerum oris is not uncommon, beginning as an ulcer on the internal surface of the cheek, surrounded by intense inflammation. Soon a black slough appears, followed by perforation. Gangrene may occur in other parts, such as the genital organs. Other complications are ophthalmia, stomatitis, rhinitis, and diarrhœa.

*Treatment.* A hot bath may be given at the onset. Remedies are directed against the bronchitis, the most useful being ipecacuanha and liq. ammon. acetat., F. 53 (General treatment, see §§ 390 *et seq.*).



§ 359. VI. **Rötheln**, or German Measles (Synon. Rubeola, Epidemic Roseola, Hybrid Measles). may be defined as an acute contagious disease, characterised by sore-throat, catarrh of the respiratory passages, and an eruption on the skin, consisting of irregular hyperæmic patches, which afterwards become confluent. Clinically, it may be said to represent a combination of measles and Scarlatina. Undoubtedly many so-called cases of Rötheln are identical with measles.

The *Symptoms* vary somewhat in different epidemics. (1) After a period of incubation, variously stated to be from 7 days to 2 or 3 weeks, the temperature rises to 100°, 101°, or 102°. This is accompanied by sore-throat and coryza. Sometimes the glands in the neck and elsewhere are swollen. When the eruption comes out the other symptoms are considerably aggravated, but the whole attack rarely lasts more than a week. The rash may be the first indication of the disease, as the primary fever is sometimes so slight. (2) The eruption appears on the 1st, 2nd or 3rd day, and consists of minute round or oval patches of hyperæmia, varying in size from a pin's head to a sixpence, very slightly raised, never papular. The rash at the outset is like that of advanced measles. In a day or two it becomes confluent, or nearly so, and the whole skin presents a scarlet hue, so that the case may be mistaken for Scarlatina. The eruption first appears on the face, and at the end of 24 hours the whole body is involved. It lasts from 2 to 8 days, and the severity of the attack is in direct ratio to the duration and severity of the eruption. It is sometimes followed by slight desquamation. The disease has to be *diagnosed from scarlatina*, in which there is no catarrh, and no "measly" eruption at the beginning of the attack. In *measles* there are no enlarged glands, no special involvement of the tonsils, but little sore-throat, and no extensive confluence of the rash. In non-specific *roseola* (rose-rash), there are no catarrh and no sore-throat.

*Etiology.* It is mainly a disease of childhood, but sometimes attacks adults. It is not so contagious as either scarlatina or measles (Murchison). One attack confers immunity.

*Prognosis.* It is a more trivial disease even than Measles, though there is a liability to some of the same complications as occur with Measles and Scarlatina (*q.v.*). The occurrence of severe bronchitis and tonsillitis are the worst symptoms we have to deal with.

The *Treatment* is like that of measles.

§ 360. VII. **Dengue** is a highly contagious fever, most commonly met with in India and America, characterised by a fugitive scarlatini-form eruption on the skin and severe articular pains. The incubation period of dengue is very short, probably a few hours or a day or two. Great epidemics occur in Africa and on the coasts of India. The fever is of a sudden onset, and ranges from 102°—105°. It is accompanied by intense headache, with extremely severe pains in the joints or limbs, much aggravated by movement. This primary fever lasts about 48 hours, and subsides by crisis. During this stage the skin may be covered with a bright red flush. During the next 1 to 2 days there is an interval of apyrexia, with freedom from pain. Then the secondary fever appears, with a return of the pains in the limbs. Both, however, are less severe than in the primary stage. A universal mottling of the skin, starting on the hands, somewhat resembling measles, though never papular, accompanies the secondary fever. As it subsides in a day or two, slight branny desquamation occurs.

*Diagnosis.* Dengue is known from *scarlet fever* by its being rarely associated with sore-throat, by the severe articular pains, and by its occurring in hot weather, and later, by its characteristic temperature. *Acute rheumatism* is rare in the tropics, has no rash, and has profuse sweats.

*Prognosis.* As regards life the prognosis is excellent; the case-mortality is extremely small. Death rarely, if ever, occurs; if so, it is from such complications as weak heart or hyperpyrexia, in the enfeebled. In most cases the patient is well in eight days. Some have painful and swollen joints for some time after the fever has gone.

The *Treatment* does not differ from the ordinary hygiene necessary in fevers. The patient should be kept in bed to avoid chill. For the pain in the limbs, belladonna, antipyrin, morphia, and in chronic cases iodide of potassium, may be given. The subsequent anæmia and enfeeblement are sometimes troublesome.

§ 361. VIII. **Typhus** (Synon. Contagious typhus. Exanthematic typhus. Germans call it Enteric Fever Typhus, Hospital, Gaol, and Ship Fever) may be defined as a contagious fever, lasting 14 days, with an eruption on the skin consisting of subcutaneous mottlings and petechial spots, with a great tendency to the typhoid state. Its disappearance from our midst is a good illustration of the triumphs of hygienic medicine. It is due to a highly contagious specific poison which can be propagated only where overcrowding, deficient ventilation, squalor, and destitution exist. As these conditions have disappeared typhus has gradually died out, though it is still occasionally met with in Glasgow, Liverpool, parts of Ireland, and other places where the poor are crowded into back-to-back houses.

*Symptoms.* 1. After an incubation period, which varies considerably, but is rarely longer than 12 days, the temperature rises rapidly for 2 or 3 days to  $103^{\circ}$ — $105^{\circ}$  or more, at which it remains until the 14th day. It starts somewhat abruptly with chilliness, rarely with rigors. There is severe headache and extreme prostration, so much so that on the second day the patient is unable to walk or stand. Drowsiness is common, and there is a typical aspect of heavy stupidity. At the end of the first week headache gives place to delirium, and this is followed by drowsiness and coma. The temperature continues to rise until the 7th day, and then falls slightly during the ensuing week, and usually ends by crisis on the 14th day. 2. The spleen is enlarged and tender. 3. The eruption appears usually on the 4th or 5th day, first on the back of the hands, arms, folds of axillæ, and in front of the chest and abdomen. It has usually two elements, which vary in their proportion: (a) subcuticular mottling, certain portions of the skin appearing hyperæmic, with fading margins. (b) reddish-brown spots, having a definite but irregular outline, varying in size from a pin's head to three lines, very slightly elevated at first, and in the course of 2 or 3 days becoming petechial, so that they will not disappear on pressure. One attack usually confers immunity. The patient ceases to be infectious the second day after the evening temperature is normal.

*Diagnosis.* 1. *Typhoid fever* was originally confused with typhus, and it is chiefly owing to the observations of Jenner that they are now differentiated. Typhoid differs from typhus in (i.) the insidious onset; (ii.) the course of the temperature; (iii.) the different eruption; and (iv.) the

diarrhœa and pea-soup stools in typhoid are characteristic. 2. In *measles* the eruption resembles the typhus spots, and appears at the same date, but in typhus it does not start on the face, it is never preceded by catarrh, is never papular, and becomes petechial. 3. Some *malarial* fevers present considerable difficulty, but they have no eruption. 4. *Uremia* and other causes of coma may be mistaken for it. 5. *Pneumonia*, meningitis, and other causes of the *typhoid state* may be confused with Typhus. 6. *Plague* sometimes presents a rash identical with typhus, but the glandular and bubonic swellings are distinctive of the former.

*Etiology.* The disease is chiefly met with in middle and advanced life. It is due to a specific contagium which has never yet been isolated. This is given off in great amount, especially during the second week, from the breath and exhalations of the patient; but the infecting distance would seem to be very short, for it is destroyed by free ventilation. Doctors frequently contract it by bending over the patient; Dr. Charles Murchison, who did so much for the study of this and other fevers, contracted typhus twice, and thus incurred the heart disease of which he died. The poison can only be produced where there is overcrowding, deficient ventilation, and personal squalor; and the strangest circumstance connected with this disease is that it cannot be developed under any other conditions, and therefore it only appears at long intervals. Thus it gave rise, with considerable colour of truth, to the theory of the origin *de novo* of infectious diseases. The malady is predisposed to by a general debility, and it is therefore commoner in times of famine and distress.

*Prognosis.* Case-mortality, 10 per cent.; between the age of 15 and 25, 4 per cent.; over 50, 50 per cent. Thus the age of the patient greatly influences the mortality. Typhus is always a serious disease, especially in the plethoric and alcoholic. It terminates fatally in three ways: (i.) degeneration of the cardiac muscle, which is a very common accompaniment of the disease; (ii.) coma, from the toxic state of the blood, or (iii.) asphyxia or hypostatic congestion of the lungs. Untoward symptoms are: (i.) weak, irregular, or intermittent pulse, or other indications of cardiac weakness; (ii.) an abundant rash, with high fever; (iii.) early and protracted cerebral signs or protracted hiccough; (iv.) all complications, especially pulmonary. Of the *complications* and *sequelæ* (i.) the pulmonary are the worst, especially broncho-pneumonia and hypostatic congestion of the lungs; œdema glottidis and pleurisy are less common. Other complications are (ii.) hyperpyrexia, meningitis, etc.; (iii.) phlegmasia and other thromboses; (iv.) gangrene of the extremities from embolism, bed-sores, and pyæmic abscesses; (v.) cardiac weakness, which may remain for a long time on account of the granular degeneration of the muscle; (vi.) post-febrile mania; and (vii.) paralysis of various parts.

*Treatment.* Hygienic treatment is essential (§§ 388 *et seq.*), especially free ventilation. Therapeutic treatment is of secondary importance. It is sometimes the practice to give an emetic at the outset. Mineral acids may assist the digestion. Stimulants in most cases are not necessary, but they must be given if the pulse is weak or irregular, or if the extremities are cold.

§ 362. **Anthrax** or Malignant Pustule (syns. Woolsorters' disease; Anthracemia; Splenic Fever—under which term the disease is registered in the Registrar-General's returns, unfortunately, because the spleen is not involved in man. Charbon, Carbunculus Vernus). The primary lesion consists of a solitary vesicle at the seat of inoculation. As the base of this becomes transformed into a central slough the contents become hardened, and around this a zone of vesicles arises. It is due to the anthrax bacillus, a relatively large organism which was one of the first to be isolated, by Pasteur.

This disease, which has a marked and prolonged vesicular stage, is most usually situated on the dorsum of the hand or arm, occasionally on the face. It affects woolsorters, furriers, felt makers, ragsorters, and others who come in contact with animals or their hides or fur; or it may be conveyed by the stings of insects. The incubation period is 24 to 72 hours. First a papule forms at the seat of inoculation, which rapidly enlarges, and becomes on the second day a vesicle, with serous or hemorrhagic contents. On the third day this bursts, leaving a raw exuding surface, which, on the fourth day, turns to a dry black slough, surrounded by a zone of intense inflammation slightly raised above the surface. Upon this inflammatory zone there appears, also on the fourth day, a characteristic ring of small red vesicles. The oedema extends around, and the lymphatics and the glands inflame. The pain is usually very slight, and no pus forms until about the tenth day, when the slough begins to separate. The constitutional symptoms vary considerably, and bear no proportion to the local mischief. The pyrexia may be so slight as not to interfere with the patient's ordinary avocation, and it may not come on until some days after the local signs; usually, however, it is severe, comes on early, and soon assumes a typhoid character.

*Diagnosis.* It may have to be diagnosed in the first phase from the sting of an insect, from various conditions which lead to solitary vesicles or bullae on the second day, from erysipelas (if on the face), lymphangitis, and other causes of oedema. The occupation of the patient assists us, but a diagnosis may be made by examining the serum or secretion of the sore, stained by Gram's method (Chapter XX.) under the microscope. The bacillus anthracis, which is the cause of the disease, is thus readily discovered.

*Prognosis.* The mortality varies with the position of the primary lesion, being 40 per cent. when this is situated on the neck or face, and 12 per cent. when situated elsewhere.

*Treatment.* The local lesion should be freely incised if seen early, and the wound irrigated continuously with carbolic lotion 1 in 20. The same substance may also with advantage be injected into the tissue surrounding the part, and repeated every 4 hours, with due care, watching for carbolic poisoning (carboloria, etc.). The patient's strength must be supported. Probably an attenuated virus will soon be procurable (§ 359).

§ 363. **Glanders** (Syn. Equinia) may be defined as a contagious febrile disease attended by a discharge from the nostrils, and sometimes an eruption on the skin, due to the inoculation of the bacillus mallei, in a person attending to horses affected with the disease. The eruption, which only occurs in ACUTE GLANDERS, consists of a general erythema, on which a crop of pustules of hemispherical shape appear in the course of a few days or hours. They vary in size between a lentil and a florin. There are also nodules of granulated material in the subcutaneous tissue and muscles, which usually suppurate, leaving large foul ulcers. The other symptoms are: (i.) a copious discharge of viscid, semi-purulent matter from the nostrils; (ii.) pains in the limbs and joints; and (iii.) high fever, with rigors and prostration, passing on to the typhoid state.

In CHRONIC GLANDERS (farcy) the pyrexia and constitutional symptoms are absent, and the cutaneous eruptions (erythema, pustules, and nodules which leave ulcers and sinuses): the discharge from the nose may be the only sign.

*Diagnosis.* The pustules of acute glanders resemble those of variola, but they are larger, and not umbilicated, and the temperature in glanders does not fall when the rash—in those cases which present a generalised pustular eruption—comes out.<sup>1</sup> The pain and swelling of the joints and limbs bear some resemblance to acute rheumatism, and still more to pyæmia.

*Treatment.* We cannot hope for much good from the treatment of ACUTE GLANDERS until such time as an attenuated toxine can be prepared. At present the disease is extremely fatal. In FARCY or CHRONIC GLANDERS the death-rate is 40 or 50 per cent.<sup>2</sup> Iodide of potash, aconite, mercury, iron, arsenic, and strychnine have all been tried, and good results have accrued from the injection of small doses of mallein.

## GROUP II. CONTINUED PYREXIA.

§ 364. In this group the pyrexia tends to assume a CONTINUED TYPE—i.e., it runs a continuous course excepting for the slight normal diurnal variation (§ 348). This group is distinguished from Group I. by the absence of an eruption, at any rate during the first four days of the illness. It is distinguished from Group III.

<sup>1</sup> The author once notified a case of this kind as small-pox, and the case passed as such through the hands of two of the most experienced medical officers of the Metropolitan Asylums Board, the mistake not being cleared up until after death, and a full investigation had been made of the circumstances under which the disease arose. It was then ascertained that the patient was a stableman attending on glandrous horses.

<sup>2</sup> Dr. Syms Woodhead in "Allbutt's System of Medicine," vol. ii., p. 524.



mainly by the course of the pyrexia ; though aberrant types of one group are found in the other.

<i>Common.</i>		<i>Rare in this Country.</i>	
I. Enteric fever . . .	§ 365	VII. Plague . . .	§ 371
II. Diphtheria . . .	§ 366	VIII. Malta Fever . . .	§ 372
III. Influenza . . .	§ 367	IX. Yellow Fever . . .	§ 373
IV. Rheumatic Fever, Pneumonia, and va- rious other Inflam- matory Disorders, usually attended by Local Signs . . .	§ 368	X. Epidemic cerebro- spinal meningitis . .	§ 374
V. Whooping Cough. . .	§ 369	XI. Relapsing fever . .	§ 375
VI. Mumps . . .	§ 370		

ENTERIC FEVER, which may be taken as a type, may present no other symptoms than *the characteristic pyrexia* ; the rash when present is ill-marked and does not appear till the second week of the disease. In DIPHTHERIA we have the characteristic *throat lesion* ; in INFLUENZA we have the *pains in the limbs* and a more sudden advent ; in PERTUSSIS, the *characteristic cough* ; and in MUMPS the *parotitis*. Various MICROBIC REACTIONS may aid us in the diagnosis. CHOLERA (§ 222) and DYSENTERY (§ 221) might also be included in this group, but the pyrexial disturbance is quite a subordinate feature compared with the intestinal manifestations.

It was formerly the custom to speak of Enteric fever, Typhus fever (Group I.), Relapsing fever and Febricula, as the "Continued Fevers of Great Britain." Of these practically only the first still prevails amongst amongst us. Febricula is generally identical with enteric. Relapsing fever has only occurred in times of famine and Typhus has disappeared with improved hygiene amongst the masses.

**§ 365. Enteric or Typhoid Fever** [Syn. Abdominal Typhus (Niemeyer), Pythogenic Fever (Murchison), Simple Continued Fever, Febricula] may be defined as an acute specific fever of about three or four weeks' duration, with a tendency to diarrhœa and the typhoid state, often attended by successive crops of rose-coloured spots ; due to a specific microbe (the typhoid bacillus of Eberth, see Coloured Plate III.), by means of which the disease is clinically recognisable. A characteristic ulceration of Peyer's patches occurs.

*Symptoms.* (1) The period of incubation is usually about 10 days, but it may be shorter or longer. The onset is extremely insidious differing in this respect from the fevers in Group I. The typical typhoid chart (Fig. 99) is the most characteristic feature of the disease ; and until the discovery of the Widal reaction we were mainly dependent upon this for the diagnosis of the

malady. In the *first* week it is "ladder-like," gradually rising with diurnal remissions until it reaches, about the end of the first week or 10 days, its highest point ( $103^{\circ}$ — $105^{\circ}$ ). During the 2nd stage, which may last a week or more, it remains continuously high, the diurnal remissions being only those that are met with in health. As the disease progresses, these daily remissions become gradually more and more marked. During defervescence, usually about the 4th week, first the morning temperature, and then the evening temperature, gradually become normal. These features are so constant as to afford a means of detecting the

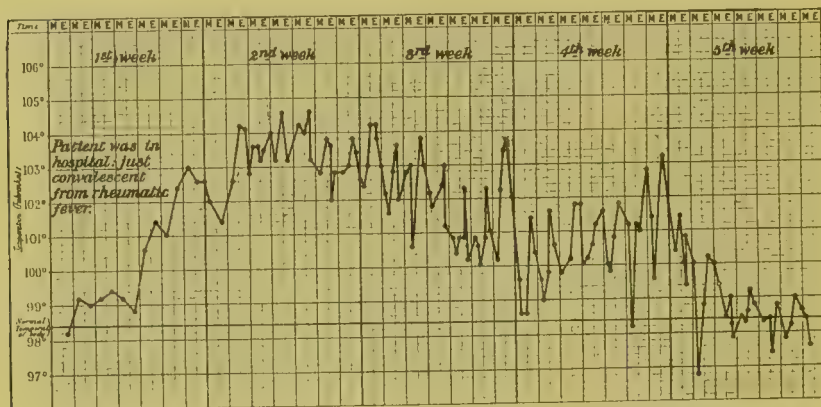


Fig. 99.—ENTERIC FEVER (typical chart). Henry H—, *æt.* 22 (under the author's care), was in hospital when he developed the enteric fever. There was apathetic mental condition, great feeling of illness and headache, watery pea-soup stools, and bronchial catarrh. The chart shows the continued character of the pyrexia in the second and third weeks, with gradually increasing remissions in the fourth and fifth weeks.

stage which a case has reached. Convalescence may be said to be established when the evening temperature has been normal for two successive nights. (2) Some diarrhœa is usually present in the early stages, and the stools are of a characteristic pea-soup or yellow ochre colour. This feature is of very little value, as a means of diagnosis, while a patient is on milk diet. In about half the cases there is no diarrhœa, and the bowels are confined. (3) The spleen is generally tender and enlarged throughout the disease, being frequently palpable even without the patient taking a long breath. Tympanitic distension of the abdomen is common, especially in the second and third weeks; and there is often pain and gurgling on pressure in the right iliac fossa, though

great care should be used in attempting to elicit this symptom, as the intestinal wall is thinned by disease. (4) The eruption generally commences to come out about the 7th—12th day (average 10th), in successive crops<sup>1</sup> of small rose-coloured lenticular spots, slightly elevated, soft, and disappearing on pressure. Each spot lasts about 3 or 4 days. They are never petechial. They are chiefly met with on the abdomen, sometimes on the rest of the trunk, very rarely on the face or limbs. The number of these spots varies considerably, but they are rarely abundant. They are very small, and may be overlooked or mistaken for flea-bites. (5) Malaise is a very constant feature from the outset, and it is for this symptom that we are generally consulted. There is rarely much pain, and no other symptom but lethargy. This is very marked, and gives rise to the aspect (*facies typhosa*), which is fairly characteristic. The lethargy gradually increases; and in severe cases the typhoid state eventually supervenes. The tongue is first covered with a thin white fur, the edges and tip being red; in the second week the fur clears off and the tongue becomes glazed and dry or red and smooth. Shallow transverse fissures are often seen on the tongue. Sordes collect on the teeth. Several *varieties* of the disease have been described, but they are not of much importance. Occasionally the disease commences quite suddenly with symptoms of great severity. The “ambulatory” form is so called because the patient is able to keep about while suffering from it. Perforative peritonitis may be its first manifestation.

*Diagnosis.* Until recently the diagnosis of typhoid was very often a matter of excluding all other possibilities, and even then was largely a matter of conjecture. But at the present time we have a valuable test in Widal’s reaction, for which purpose a specimen of the patient’s blood must be procured (§ 351) and sent to a laboratory. The diazo test is also of service, though not so certain. Undoubtedly many slight cases of typhoid are overlooked or spoken of as *Febricula* (*vide infra*). Slight cases are also apt to be mistaken for *Influenza*, which, excepting for the pulmonary symptoms, the more sudden advent, and brief duration, much

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<sup>1</sup> This fact may be revealed by enclosing each of the spots which appear on one day by a circle, next day by a triangle, and so on, by a nitrate of silver paint or aniline ink.

resembles mild typhoid. The other *specific fevers* in this group may also have to be excluded. In most cases of typhoid there appears early in the disease a generalised *bronchial catarrh* and *hypostatic congestion* of the lungs, and nothing is commoner than to mistake enteric fever, in its early stages, for pulmonary congestion or bronchitis, and severe cases may be mistaken for *pneumonia*. These pulmonary disorders should be recognised by the relative absence of the prostration, and the diarrhœa, enlarged spleen, etc., of enteric. In severe cases of typhoid, early delirium may occur and suggest *meningitis*; but the latter is recognised by (i.) the retracted abdomen; (ii.) the irregular and sighing respiration appearing early in the disease; and (iii.) the headache persists longer, and may concur instead of alternating with the delirium (Murchison); signs of intracranial pressure also supervene, such as ptosis, squint, optic neuritis, and other local paralyses. *Acute Miliary tuberculosis* is a disease which sometimes so closely resembles enteric, that, as Niemeyer<sup>1</sup> remarks, they can only be differentiated in the dead-house. The positive signs of typhoid are wanting; and the presence of tubercle is suggested by—(i.) the intermittent character of the temperature and its prolonged course; (ii.) the lung symptoms are much more marked; (iii.) the rapidity of the breathing is out of proportion to the other signs of illness; and (iv.) the pallor and lividity of the face and the rapid emaciation are also more prominent features. *Malignant endocarditis* is recognised by (i.) the intermittent character of the temperature (usually), and (ii.) the cardiac signs. *Pyæmia* is differentiated by the wide range and irregularity of the pyrexia (§ 384).

*Etiology.* Enteric fever is now known to be due to a specific microbe which has been isolated. *All matters which the patient discharges from his stomach, bowels, and bladder are infective.* Most epidemics are due to the contamination of the water supply by sewage. The disease has also been traced to the eating of oysters,<sup>2</sup> to ice-creams, and to the milk supply. To produce the malady the microbe must be introduced into the alimentary canal; thus, nurses and friends contract the disease in handling

<sup>1</sup> "Text-book of Practical Medicine." This was before the discovery of bacillus and Vidal's reaction.

<sup>2</sup> Sir William Broadbent.



the stools and sheets, or any other articles which have been contaminated by the feces and urine. The excreta become more virulent after standing from 12 to 24 hours. The malady is most prevalent in the autumn and early winter; and Pettenköfer has found by several years' observations that typhoid outbreaks are favoured by (i.) a rapid falling (after a rise) of ground water, that is to say, a well aerated moist soil; (ii.) a certain temperature of the earth; and (iii.) pollution of the soil by animal impurities. One attack does not necessarily confer immunity, as second attacks are not very uncommon. The malady is chiefly met with in young people between 10 and 30 years of age.

*Prognosis.* The case-mortality varies in different epidemics from 15 to 20 per cent. The prognosis is more favourable in the young. It is always a serious disease on account of the numerous complications, prolonged course, and its exhausting nature. The usual duration is about 3 or 4 weeks, though it varies from 10 days to 6 weeks even without relapses, which are by no means infrequent. *Untoward symptoms.* The height and the continued character of the fever are the best guides to the severity of the attack. Many of the fatal issues would be avoided if it were remembered that slight attacks require just as much care as severe ones, being liable to be attended by hæmorrhage and perforation if the patient does not remain at rest. The prognosis is grave when the fever remains at about  $104^{\circ}$  throughout the 2nd week, and especially if the diurnal remissions do not increase as they should do in the 3rd week; it is also grave when there is vomiting, excepting at an early stage, urgent diarrhœa at any time, severe tympanitis, or hæmorrhage. A sudden fall in the temperature suggests hæmorrhage or the occurrence of peritonitis. The most common *complications* are (1) those of the lungs, and, as previously mentioned, bronchial catarrh and hypostatic congestion are practically symptoms of the disease. Pneumonia and pleurisy also occur. (2) Hæmorrhage, due to the ulceration of Peyer's patches, occurs in 8 or 10 per cent. of the cases. (3) Peritonitis, due either to the spread from the ulceration or to perforation, is a frequent complication, and it is sometimes peculiar in being latent, that is to say, unattended by the pain which is so characteristic of

that disorder. Its occurrence can then only be recognised by (i.) vomiting; (ii.) great aggravation of the already existing prostration; (iii.) a small rapid pulse (120—140); (iv.) immobility and distension of the abdominal walls; and (v.) a sudden fall of the temperature. (4) Other complications are thrombosis of the femoral vein, local suppurations and inflammations, such as parotitis, periostitis, cancrum oris, and laryngeal ulceration. As *sequelæ*, dementia, peripheral neuritis, phthisis, and miliary tuberculosis may occur.

*Treatment.* The microbe of enteric fever, by virtue of its special "proclivity," attacks Peyer's patches in the small intestine, which become inflamed, swollen, and ulcerated. Consequently, there are three indications: (a) to prevent peritonitis, hæmorrhage, or perforation by rest and suitable diet; (b) to maintain the strength of the patient; and (c) to neutralise the toxin of the microbe (by serumtherapy, see § 389). Absolute rest is of the highest importance, and when the diagnosis has become established the patient should not be allowed to turn in bed. Grave responsibility rests upon the nurse in this respect, for perforation may occur in changing the draw-sheet. Diet is also of prime importance. Milk is the staple article, and not less than  $2\frac{1}{2}$  pints a day should be given; it is advisable to add barley water or lime water to prevent the formation of large curds. The addition of three grains each of sod. bicarb., mag. carb., and sod. chloride to a cup of milk has a like result. If milk disagrees, give whey, egg albumen, or butter-milk. Clear soup, chicken broth, and beef-tea may also be given. No solids should be taken on any account. Predigested foods are of great aid to promote assimilation, especially if the tongue be heavily furred, and pepsin is said to have quite a specific effect on the disease, though it probably acts in that way. It may be given thus: essence of pepsin,  $\mathfrak{m}$  xxx., acidi nitrohydroch. dil.  $\mathfrak{m}$  v., glycerini, ad  $\mathfrak{z}$ j. The bowels must be regulated. Some say the diarrhœa should be encouraged; but if profuse it weakens the patient, and must be checked by enemata of starch and opium (3ss. of tinct. opii. to  $\mathfrak{z}$ iii. of mucilage of starch); or liq. morph.  $\mathfrak{m}$  xx. with acid sulph. dil.  $\mathfrak{m}$  x., every 3 or 4 hours. If this fail, give acetate of lead, bis. carb. or bis. salicylate. On

the other hand, the bowels must be opened at least every third day; some administer calomel, but it is better to use an enema of soap and water. If peritonitis supervenes (*vide supra*) apply heat to the abdomen, give opium in large and frequent doses, iced milk, and very small doses of brandy or champagne. If the abdomen is tympanitic apply turpentine fomentations and give turpentine internally. Hæmorrhage should be checked by the administration of opium, and absolute rest enjoyed. To maintain the strength stimulants are generally called for, but they should not be given as a matter of routine; *the pulse is the best indication* for their administration; they must be stopped if hæmorrhage occur.

*Prophylactic treatment* is based upon a knowledge of the origin of the disease, and the mode of its introduction into the system—viz., by the mouth (see § 391). Preventive and remedial inoculation are now on their trial (see §§ 388 *et seq.*), but there is great promise of success.<sup>1</sup>

**Febricula** is a term somewhat loosely applied to any condition which is chiefly evidenced by a slight degree of fever and malaise. It was formerly classed as one of the four Continued Fevers of Great Britain. Murchison<sup>2</sup> showed that when it assumed the form of a definite illness it was in reality a mild attack of typhoid fever; and that when death, which was a very rare event, did ensue, the characteristic lesions of that disease were found in the intestines.

§ 366. **Diphtheria** (Syn. Membranous Croup<sup>3</sup>) is a contagious fever, characterised by a membranous exudation on the fauces, due to the Klebs-Löffler bacillus (Chapter XX.). *Symptoms.* The incubation period is uncertain, but it is said to be about 2 to 6 days. (1) The onset is usually gradual (extending over a day or two), but in some cases it is sudden. The fever is often high, but in others it may not exceed 102·5°, and it may even be quite normal (Bristowe) in asthenic cases. The height of the temperature is no guide to the severity of the disease. *The temperature chart does not conform to a regular type*, but Fig. 100 represents a common case. (2) Sore-throat is present from the beginning, and frequently dysphagia. On one or both of the tonsils there is a characteristic patch of creamy white, “wash-leather” like

<sup>1</sup> Professor A. E. Wright: *The Lancet*, March, 1901.

<sup>2</sup> “The Continued Fevers of Great Britain.” Murchison, 2nd Ed., 1873.

<sup>3</sup> Diphtheria was probably included under the term Malignant Sore-throat or Cynanche Malligna prior to the year 1855 (about).

membrane surrounded by a red areola. If removed, this leaves bleeding points. As the patches extend they run together and may spread on to the soft palate and uvula. Their occurrence on the soft palate is a diagnostic feature of great value from quinsy. The patient complains that the neck feels very stiff, and the glands at the angle of the jaw are swollen. This glandular enlargement dates from, or even before, the recognition of the

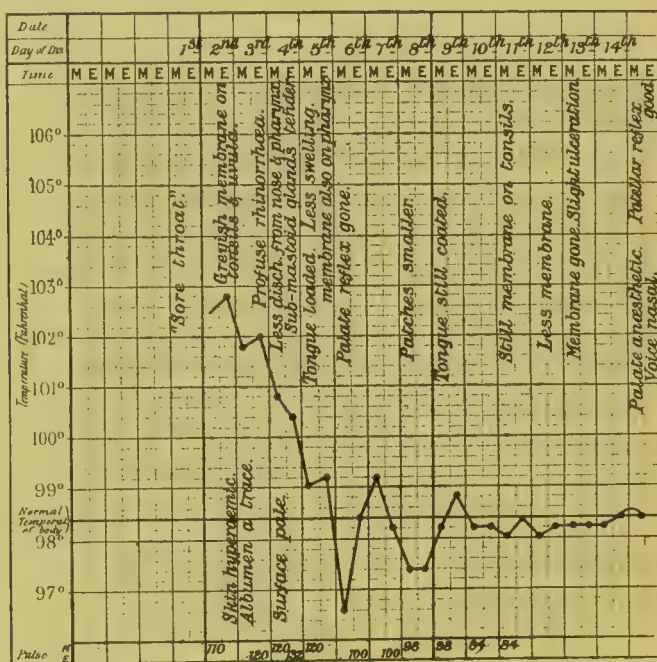


Fig. 100.—DIPHTHERIA. Male, æt. 9. An ordinary case of faucial diphtheria without implication of larynx. The palate was still anæsthetic one month later. Not followed by paralysis. The different events are indicated on the chart, for which the author is indebted to Dr. F. F. Caiger.

attack, and is of importance in the diagnosis. The membrane spreads to the larynx and bronchi in grave cases, and it may also spread upwards to the nose (it often does in children). An ichorous discharge from the nostrils in a child lying prostrate and fretful in bed is very characteristic of diphtheria. It may, in rare cases, involve, or start upon the conjunctivæ, genitals, or on the skin at the angles of the mucous orifices. (3) Albuminuria is present in about half the cases (Caiger) towards the end of the



first week. There may be hyaline and epithelial casts in, sometimes suppression of, the urine. (4) Prostration and anæmia are very marked, but the mind usually remains clear to the end, even in lethal attacks. In the asthenic type of the disease lassitude and prostration are extreme. Eruptions on the skin are occasionally met with, the commonest being an erythema, or purpuric spots in fatal cases.

The *Diagnosis* of diphtheria may be made by finding the Klebs-Löffler bacillus in swabbings taken from the seat of the disease; but, since there are several pseudo-diphtheria organisms, and since its cultivation requires several days, it does not do to put too much faith in the bacteriological examination, unsupported by experiments on animals.<sup>1</sup> The diagnosis of the sore-throat caused by tonsillitis, scarlatina and diphtheria presents certain difficulties, and is given in the tabular form (§ 114). *Follicular tonsillitis* is distinguished by the absence of the wash-leather-like patches on the tonsils, nose, or larynx, and the presence of higher fever. There may also be a history of previous attacks. *Scarlatina* is distinguished by its abrupt onset, its higher fever, its rash, strawberry tongue, and generally the absence of membrane from the throat. Simple "*croup*" (catarrhal laryngitis) is distinguished by the absence of patches in the throat, and the absence of albuminuria. *Membranous croup* is always diphtheric.

*Etiology.* The disease occurs chiefly in the young and especially under 10 years of age. It is also predisposed to by measles, whooping cough, and other infectious fevers. There seems to be a more marked tendency in certain families to contract this disease than is the case with other infectious maladies. The disease spreads from person to person; it may be conveyed by instruments, infected handkerchiefs, kissing, etc., and it *hangs about a house or district with remarkable tenacity*. Nurses and medical men frequently contract the disease by the patient coughing into their faces. It may also be conveyed by milk; but there is no evidence that it is conveyed by water. Some hold that the disease is predisposed to by bad air from drains, and undoubtedly a form of sore-throat may be thus developed. It is probable, however, that these conditions only favour

<sup>1</sup> See also Dr. H. Donkin, "The Diagnosis of Diphtheria": *Brit. Med. Journ.*, Nov. 3, 1900.

the development of the diphtheritic poison. Human beings may contract the disease from cats, and epidemics have been produced in this way.

*Prognosis.* The case-mortality varied widely in different epidemics, but it used to be an average from 25 to 50 per cent. Since the introduction of the serum treatment the mortality has fallen to approximately 10 per cent. (Caiger). The malady is mostly fatal by the spread of the membrane to the larynx during the first week of the disease. After the first week death may take place by asthenia (from the toxic state of the blood), syncope, or complications. Pharyngeal cases are, in adults, usually mild, and recover in a week or so, but severe cases last 2 or 3 weeks. Great care is required even in the mildest cases, lest the membrane should spread, and on account of the complications (*q.v.*). The clinical varieties according to Caiger (*loc. cit.*), are—(1) *Mild faucial* cases, mostly met with in adults; (2) *Severe faucial* cases, with a tendency to extension, chiefly met with in young children; (3) “*Croup*” or *laryngeal* diphtheria, where the air passages are alone affected; (4) *Nasal diphtheria*, where the nasal passages alone are affected; (5) Diphtheria of *other parts*—cheeks, gums, tongue, lips, conjunctivæ, genitals, wounds, etc.—conditions generally associated with faucial or laryngeal diphtheria. *Untoward symptoms.* The prognosis is unfavourable when the temperature is low in spite of severe local lesions; when epistaxis or any form of hæmorrhage occurs, or there is much albumen in, or suppression of, the urine; and when the patient is young (see above). Rapid extension of the membrane is also a grave sign, especially when it extends down the larynx, leading to croupy cough, dyspnœa, and cyanosis; and death takes place in such cases from asphyxia, unless they are promptly relieved (see below). The chief danger in the second week is cardiac dilatation and failure; and the pulse and heart should be closely watched at this time. Of the *complications*, certainly the commonest is paralysis, due to peripheral neuritis. It attacks nearly 20 per cent. of the cases (Caiger), and comes on usually about the fourth week; sometimes later. The characteristics of diphtheritic paralysis are: (i.) it starts usually in the palate; and therefore nasal voice or dysphagia is the earliest symptom, and fluids taken are returned through the nose. The

paralysis is progressive, and tends to involve many, sometimes all, of the muscles of the body. Next in order we may get—loss of accommodation, squint, loss of patella reflexes. The diaphragm and intercostals are amongst the most serious. (ii.) Motion and sensation are simultaneously affected, though often the sensory symptoms are the first to be observed. The attitude assumed in marked cases is very characteristic—the little patient shambles into the room with drooping shoulders and head bent forward from weakness of the neck muscles. (iii.) In general terms there is a tendency to complete recovery in a few weeks, though sometimes death occurs apparently from involvement of the vagus. (2) Broncho-pneumonia, so frequent formerly, only attacks about 4 per cent. under modern methods of treatment; but cardiac dilatation, probably due to myocarditis, is a frequent occurrence. (3) Nephritis and dropsy during convalescence are very infrequent, and permanent lesions of the kidney are rare. Otitis media is not uncommon.

*Treatment.* The indications are (a) to neutralise the toxin in the blood; (b) to inhibit the local process; and (c) to strengthen the constitution to resist the disease. (1) Thanks to the recent advances in science we now have a powerful *antitoxin* for the control of the disease, and if given early it is capable of completely neutralising the toxin and arresting the disease. It is a good general rule to give it in all cases, though there are three possible exceptions<sup>1</sup>—viz., (i.) very slight cases, when under constant medical observation; (ii.) cases seen too late in the course of the disease, and the membrane is obviously separating (the antitoxin must be given at the earliest possible moment, doses and methods are given §§ 388 *et seq.*); and (iii.) cases where there is marked irregularity and feebleness of the heart, which require caution. (2) For the *local* treatment, nitrate of silver, hydrochloric acid, and other caustics have been much used, but are now condemned as useless and harmful; and papain, with borax, had a reputation which no longer exists. Disinfectants are certainly useful, applied by syringing, or spraying, or swabbing every hour 1 in 10,000 corrosive sublimate, or carbolic acid;  $\frac{1}{2}$  per cent. are not without danger; but the best results have

<sup>1</sup> See also Washbourn, *Lancet*, October 14, 1899, p. 1019.

been obtained with chlorine (F. 18), formalin  $\frac{1}{2}$  per cent., chinosol  $\frac{1}{6}$  per cent., or sulphurous acid used by syringing or spraying. Warm inhalations every half-hour and hot applications to the neck give much relief. When the nose is affected it must be syringed with the same disinfectant solutions. When the larynx is much affected, the question of tracheotomy has to be considered. Statistics used to be very unfavourable, the mortality being 70 or 80 per cent. But in the present day over 70 per cent. of the cases operated on recover; and tracheotomy should be performed promptly whenever the breathing is difficult owing to laryngeal obstruction. The results are more satisfactory when it is *done early*; and all laryngeal cases should be closely watched for the epigastric retraction during inspiration which indicates inspiratory dyspnoea. It is then essential to keep the patient in a steam tent. (3) The *constitutional* treatment consists of stimulating and supporting treatment. In asthenic cases ferri. perchlor. and pot. chlor. are generally given, with wine, beef-tea, and abundant nutriment. In the inflammatory forms give diuretics and pot. chlor., with salines and laxatives. In all cases the patient should be kept quite still in the recumbent position for fear of the heart failure, which is apt to occur, especially about the 10th to the 21st day.

§ 367. **Influenza** is an epidemic fever attended by considerable prostration and usually by catarrh, and a tendency to the development of local inflammations. It has been known for at least five centuries, and has occurred at various times in great epidemics, separated sometimes by many years' interval.

*Symptoms.* (1) After an incubation period of 1 to 6 days the patient's temperature goes up in the course of a few hours to  $102^{\circ}$  and  $104^{\circ}$ . The onset is frequently attended by shivering. The fever generally ends in 1 to 5 days with profuse perspiration. The pyrexia is attended more or less by pains in the limbs, and it is these pains in the limbs which form such a characteristic feature of influenza. (2) "Catarrh" usually attends the fever—*i.e.*, there are redness and watering of the eyes, running at the nose, sore-throat, sneezing, tightness of the chest, etc. (3) Malaise and prostration out of proportion to the amount of pyrexia attend the fever. (4) Some cases have only the three symptoms just



mentioned, but there is a great tendency to local complications. The *type* of the disease therefore varies according to the physiological system mainly involved. (i.) The *respiratory* tract is very frequently attacked, and in that case bronchitis and pneumonia complicate the disease. (ii.) The *circulatory* system may be attacked by endarteritis, and occasionally, but not often, other gross lesions. The neuro-vascular apparatus is however specially prone to suffer, causing tachycardia and bradycardia, palpitation, flushings, faintings, perspiration, dyspnoea, and the like. (iii.) Involvement of the *alimentary* tract may be evidenced by gastro-enteritis, diarrhoea, vomiting, jaundice, etc. (iv.) *Eruptions* on the skin may occur, especially urticaria, erythema, or rose spots like measles. (v.) The *nervous* system, especially in the aged, is affected for long after the disease, and neurasthenia is particularly apt to supervene. Peripheral neuritis is frequent. Depression, prolonged mental dulness, and other symptoms are met with.

The *diagnosis* is not difficult in typical cases, especially when the disease is prevalent. The short duration of the initial symptoms and the usual absence of rash are sufficiently characteristic. The severe pains in the limbs are very typical.

The *etiology* of the disease is still obscure, though a specific microbe has been isolated, which occurs chiefly in the secretion of the respiratory tract. It is certainly epidemic, but it is still doubtful whether it is contagious in the true sense. It appears to break out in several persons spontaneously in certain localities. One attack confers no immunity from a second. As regards predisposing causes, age has no influence, nor have seasons of the year, nor sanitary conditions. Old and young, rich and poor, all are attacked alike.

*Prognosis.* The case-mortality is about 1 per cent. among the old and young together. In middle-aged and elderly people the respiratory type is very apt to end fatally with pneumonia, and undoubtedly many cases presumed to be primary pneumonia are really secondary to this disease. It is fatal only through its complications. The disease itself is usually trivial, and the patient recovers in the course of a few days. Relapses are not infrequent. The *complications* consist of those mentioned above under

types of the disease. The *sequelæ* are prolonged weakness, peripheral neuritis (sometimes attended by tremors), otitis, orchitis, meningitis, and mental derangement.

*Treatment.* During the attack the patient should be kept in bed in view of the complications and sequelæ. Sod. salicyl., antipyrin, and antifebrin will reduce the fever and relieve the pains in the limbs; and ammoniated tincture of quinine is a justly popular remedy. For the rapid heart liq. arsen. is recommended (m iv. t.d.s.).<sup>1</sup> It is well to keep elderly people indoors during the prevalence of the disease, as they run greater risks from its effects.

§ 368. **Rheumatic fever, Pneumonia, and other inflammatory disorders**, which usually present well-marked **local manifestations**. The three fevers just described are those most commonly met with in England, in which the pyrexia may run a continued course, and which have no eruption during the first 4 days. But it must not be forgotten that certain inflammatory disorders may give rise to pyrexia of a continuous type, and that the usual local signs of these disorders may, at the time when the patient is first seen, be absent. It will be well, therefore, to mention those which might be mistaken for an acute specific fever.

(a) **OBSCURE (so-called) LOCAL<sup>2</sup> INFLAMMATORY DISEASES** are mostly met with as complications secondary to fevers. They can usually be detected by a thorough examination of all the organs in the body (§ 350). Nevertheless, certain cases of (1) *pericarditis*, or (2) *pneumonia* or *pleurisy*, may be latent—i.e., the usual physical signs may occasionally be wanting or overlooked. (3) Various affections in or around the *throat and nose*; (4) some *abdominal* disorders, such as perihepatitis, inflammation of the mesenteric glands or pancreas, etc.; (5) certain rare cases of *sarcoma* and *carcinoma*; or (6) inflammation of the *meninges*, tubercular or epidemic, may also give rise to an elevation of temperature sometimes unattended by marked local symptoms.

(b) Certain obscure **GENERAL INFLAMMATORY DISORDERS** are

<sup>1</sup> "Effects of Influenza on Heart and Circulation." Sansom. *Lancet*, October 21, 1899, p. 1075.

<sup>2</sup> The word local is here used in a qualified sense; compare for example footnote p. 185.

attended by pyrexia, which may similarly give rise to difficulties in diagnosis. (1) In *rheumatic fever* and *acute gout* the pyrexia is nearly always continuous. The joint lesions are the cardinal feature in these cases; but it must not be forgotten that acute rheumatism may commence with inflammation of the pericardium (the structure of which very much resembles that of a joint), and that the joint lesions may not be apparent for several days. (2) There are several conditions special to infancy and childhood which are attended by continued pyrexia—(i.) *infantile paralysis* (acute anterior poliomyelitis) is attended at its outset by a considerable rise in temperature, which may last for several days or weeks, and be accompanied by restlessness, peevishness, etc. (ii.) *Rickets* from time to time may have a slight degree of fever, accompanied by a generalised tenderness and profuse perspiration; and, as just mentioned, (iii.) *meningitis*, tubercular or epidemic. (3) A *nervous or hysterical pyrexia* has been described, and I have seen the temperature go up in an erratic manner, at odd times, in nervous subjects. But while admitting that the nervous system plays a very important part in the production of fever (as witness the rigors and pyrexia which follow catheterisation), it is difficult to believe that there is not a compound cause in operation in such cases. Only a thorough *post-mortem* and bacteriological examination would enable us to be certain that none of the many obscure foci of inflammation above mentioned were present.

§ 369. **Whooping Cough** (Pertussis) is an acute specific infectious malady, characterised by paroxysmal attacks of coughing followed by a long noisy inspiration (the whoop). The period of incubation is from 3 to 14 days (average five). (1) The onset is marked by a preliminary catarrh, or running from the nose and sometimes the eyes, attended not infrequently by paroxysmal dyspnoea and drowsiness. This premonitory stage lasts only for 24 or 48 hours, and may be overlooked. (2) Paroxysms of coughing then set in. Each paroxysm consists of a series of short sharp coughs, followed by a *loud inspiratory "crow,"* through the narrow chink of the half-closed glottis, and it is often followed by vomiting—a diagnostic feature of value when we have to depend on the mother's account of the case. After some days the face remains somewhat swollen, and subconjunctival hemorrhages may also occur. There are no physical signs characteristic of the malady, unless, as some maintain, enlarged bronchial glands can be detected by percussion or ausculto-percussion over the root of the lung. Bronchitic sounds are generally present in greater or less degree. (3) The constitutional symptoms vary

considerably in severity. In many cases they are absent, the temperature being hardly elevated and the child being apparently quite well between the attacks of coughing. In typical cases, however, there is slight pyrexia. The *diagnosis* is not difficult, since the paroxysms of coughing are very characteristic.

*Prognosis.* All the symptoms increase for the first ten days, then remain stationary for a few days, and decline during the ensuing week. It is usually one of the trivial ailments of childhood. It is severe only in very young children, in the weakly and the rachitic, or by reason of its complications, of which there are three chief ones, viz.: bronchitis, broncho-pneumonia and convulsions. Ulceration of the frenum of the tongue is common, due to the forced protrusion against the teeth in the act of coughing.

*Treatment.* In view of the fact, which does not seem to be sufficiently known, that children living near gas works and bleaching works do not get the disease, it would be worth while to try inhalations of coal tar. Belladonna is, in my experience, the most useful amongst the drugs, though nothing seems to cut short the malady. It should be given in large doses; children will stand 10 to 20 minims of the tincture. Antipyrin, hydrocyanic acid, carbonate of ammonia, ipecacuanha wine, conium, and the bromides have also been recommended. How long a child remains infectious is an important practical question. Infection lasts as long as the characteristic cough is present, but when, as in some cases, it is hard to say whether the attacks are typical or not, it is best to take three to four weeks from the commencement of the disease as the duration of the infection.

§ 370. **Mumps (Acute Parotitis)** is an acute febrile infectious disorder characterised by inflammatory swelling of one or both parotid glands. The period of incubation is from 1 to 3 weeks.

The differential *symptoms* consist of moderate fever ( $102^{\circ}$ ) subsiding in the course of a week, with stiffness of the jaw, and difficulty of swallowing, due to swelling and inflammation of the parotid gland. Sometimes the submaxillary and sublingual glands are also involved. The glands may swell so as to completely prevent the patient opening his mouth more than a quarter of an inch. They are acutely tender, and disfigure the patient very much, but the malady is essentially a trivial one. The *diagnosis* is very simple, the swelling of the glands being unlike anything else. *Etiology.* It is almost entirely confined to children and young persons between the ages of 5 and 20. It is rare in the very young and very old, but is often epidemic, and runs through a school. A patient remains infectious as long as there is any definite swelling of the glands. *Prognosis.* Death from the disease is unknown; and the patient is generally quite well in 10 or 12 days at the outside. The chief danger is the swelling of the tonsils and submaxillary glands. In scrofulous subjects the swelling is slow to disappear. Suppuration of the infected glands is very rare, excepting in the parotitis which accompanies typhus or some other infectious fever. The *complications* consist of (1) enlargement of the tonsils, and (2) orchitis and ovaritis. In these circumstances a very curious phenomenon occurs, for as the testis swells, the parotitis subsides. It is the best instance of the phenomenon called "metastasis." The mammary glands may also become swollen and tender. In some epidemics the swelling of the mamma or testicle precedes or



accompanies that of the parotid; and epidemics have been known in which the former were involved without any parotitis. Occasionally the affected glands may become permanently atrophied. *Treatment.* The patient should be kept in one room. Warm anodyne fomentations may be applied; and if tension is present, leeches give relief. Diaphoretics and purgatives are useful, and nutrient enemata may be required.

*The remaining fevers in this group are* PLAGUE, YELLOW FEVER, MALTA FEVER, *which are met with abroad*; RELAPSING FEVER, *met with only in times of famine*; and EPIDEMIC CEREBRO-SPINAL MENINGITIS, *which is very rare in this country.* In HAY FEVER, DYSENTERY, and CHOLERA, *there is some disturbance of the temperature readings.*

§ 371. **Plague** (*Pestes major, Bubonic plague, Typhus Bubonicus, Oriental plague, probably the same as the Black Death*) may be defined as a highly contagious and fatal fever, characterised by inflammatory, glandular, and peri-glandular swellings, hæmorrhages beneath the skin and from the mucous membranes, and the typhoid state. The last great epidemic in London was in 1666. Its endemic centres in the present day are Northern India, Yunna (China), Mongolia, and possibly Uganda. Since 1894 many small epidemics of plague have arisen in several different places.

*Symptoms.* (1) The incubation period is from 2 to 8 or even 15 days (Manson). (1) There is often a prodromal stage with depression and pains, but usually the onset is sudden, with shivering, and fever rising to 103° or even 107°. Mental aberration is not uncommon. The prostration is very marked, and may be accompanied by vertigo, staggering gait and lethargy, soon passing into the typhoid state. The spleen and liver are usually enlarged. In some cases the speech is halting and staccato, the expression vacant, and the eyes congested. (2) Buboes (inflamed glands) appear in 1 to 5 days, usually within 24 hours. They may be single, or a group may be involved in one place, usually the groin; sometimes they appear in several parts of the body at once. They may be painless or very painful; and they may suppurate about the 7th day. (3) Petechiæ and subcutaneous hæmorrhages are not uncommon. A distinctive rash is rare, but when present it resembles typhus. There are six principal *varieties*, which prevail in different epidemics—(i.) The *bubonic* variety is the commonest: glandular swellings occurring in quite 70 per cent. of all the cases<sup>1</sup>; (ii.) the *septicæmic* type is very fatal; the glands enlarge slightly, but they do not suppurate; (iii.) an *abortive* form, in which there are buboes without much fever, subsiding in 14 days; (iv.) a *fulminant* form, with high fever, little glandular enlargement, vomiting of blood and death within a few hours; (v.) a *pneumonic* form, which may be mistaken for bronchitis or pneumonia, attended by intense prostration, no glandular enlargement, and death usually on the 5th day; the pulse-respiration ratio being not so much altered as in true pneumonia; and (vi.) an *ambulant* or mild form with chronic glandular enlargement, great anæmia and weakness. The *diagnosis* is not difficult in presence of sudden onset, marked prostration, mental state, and bubonic swellings. Plague closely resembles typhus both in its symptoms and etiology, but a rash is rare in the former; and the microbe

<sup>1</sup> Mr. Jas. Cantlie's description of the malady issued by the London County Council (October, 1900); to which the author is also indebted for other items of information.

of plague is distinctive.<sup>1</sup> Enteric fever may have to be diagnosed from Plague, though its onset is so much more gradual.<sup>2</sup>

*Etiology.*—Plague is due to the bacillus pestis, discovered first by Kitasato, and later by Yersin. It can be readily demonstrated in the serum drawn from the periglandular tissues by a hypodermic syringe. It was observed that outbreaks of plague were often preceded by a large mortality among rats and other vermin, and it is now believed that the disease is mainly conveyed by rats. Filth and overcrowding predispose to plague. Age and sex have little influence. The disease is highly contagious, but "its extension depends more on place infection than on direct transmission from person to person" (Manson).

*Prognosis.* The case-mortality in the early periods of epidemics is generally 100 per cent., later on 40 per cent. Amongst the white population in India it was only 35 per cent. (Cantlie). In the usual course, death occurs before the 6th day, or if the patient is to recover, convalescence starts between the 6th and 10th day. Prolonged suppuration of the glands may delay convalescence considerably. The course of the disease is very difficult to forecast. Hæmorrhages usually herald death. The *sequelæ* consist of boils, pneumonia, dropsy, partial paralysis, and mental disorder.

*Treatment.* The hygienic and therapeutic treatment are as in typhus (see also §§ 388 *et seq.*). The injection of carbolic acid into the glands has been practised with some success, and large doses by the mouth are also recommended. Immunisation is now obtained by inoculation of serum (Yersin) if commenced early in the course of the disease.

§ 372. **Malta fever** (syns. Mediterranean fever, Gibraltar fever, Febris Undulans) arises only in the countries around the Mediterranean Sea, chiefly in the summer months, but it recurs in England after persons have left those districts. It is predisposed to by bad drainage and unhygienic conditions. It is now known to be due to a micrococcus which has been found in the blood. It has an insidious onset, with languor and increasing debility. The temperature stands about 102° to 104° for a week or a fortnight, then it falls. In mild cases this may be all; but more often, after an interval of a week or so, the fever returns; then it falls again. The pyrexia runs a most indefinite course, neither continuous nor intermittent, though it may be either. In two cases I have recently seen the temperature resembled pyæmia more than anything else. The most characteristic feature of this disease, however, consists in the *attacks of pyrexia with marked prostration, separated by intervals of comparative health.* These attacks may be repeated over several months. The spleen becomes enlarged, and there is usually a certain amount of gastro-intestinal disturbance. Among the symptoms complained of are rheumatic pains and sometimes swelling in the joints and limbs. The weakness may amount almost to paralysis. Thrombosis is fairly common.

*Diagnosis.* This fever is very apt to be mistaken for *enteric fever*, as the gradual onset is common to both. But in typical cases of enteric, there is a more prolonged and characteristic course, and the Widal reaction

<sup>1</sup> The Code of regulations for searchers of the plague issued by the Royal College of Physicians in 1665, mentioned a rash as a means of recognising the disease, but probably typhus was confused with plague. Murchison held they were probably identical, but the chief fact which disproved this was their seasonal incidence. Plague was a summer disease most prevalent in July and August. Typhus was most prevalent in the winter.

<sup>2</sup> See also *The Lancet*, Oct. 27, 1900, p. 1197.

(*q.v.*), which is obtainable in both diseases, affords a certain means of distinction; each disease having its own characteristic serum reaction (Prof. A. E. Wright). Some cases may be mistaken for *malaria*, but the malarial parasite is not found in the blood.

The *prognosis* as regards life is good. The case-mortality of Malta fever is only 2 to 3 per cent. But when a patient is attacked with Malta fever at the beginning of the summer months, and cannot be removed from the Mediterranean at once, he may have a long series of relapses, with consequent anæmia of great intensity. So long as the tongue remains coated a relapse may be feared. The usual complications are excessive sweating, orchitis, diarrhœa, and cardiac failure. The most severe complications, however, are pneumonia or "abscess of the lung," and hyperpyrexia; the latter being the usual cause of death.

*Treatment.* It is most important, if the disease occurs in the beginning of the summer, to immediately remove the patient to a cooler climate. As regards drugs, they should be avoided. Antipyretics are dangerous; if the fever is high cold sponging is preferable (see § 393). Professor A. E. Wright, of Netley, has prepared an antibacterial toxin.

§ 373. **Yellow Fever** is an acute specific fever peculiar to hot climates and seaport towns, accompanied by jaundice, black-vomit, and the typhoid state.

*Symptoms.* (1) The incubation period is short, probably from 4 to 5 days. One attack usually renders the patient immune for life. Yellow fever has a sudden onset, the temperature rising on the first day to 101°, 105°, or even higher, and it remains high for three or four days. The pulse does not rise in proportion. The temperature then falls to normal, or at least remits greatly; and though it may rise again, the second fever is not so high. (2) Albuminuria is a pathognomonic sign, and occurs usually in 18 to 24 hours after onset. The urine often has a green colour. (3) In most cases jaundice and vomiting appear about the second day; but in mild cases these may be absent, so also the yellowness which has given the fever its name. In severe cases the jaundice is intense, with petechiæ, the vomit is mixed with bile, and in the later stages, with blood, forming the "black vomit." Hamorrhages may also occur from the gums, stomach, nose, and bowels. There is no splenic enlargement. The liver has a degree of fatty degeneration, and the blood vessels supplying the stomach and intestines are in a state of degeneration, and readily rupture.

*Diagnosis.* Yellow fever has to be diagnosed from the pernicious forms of *malaria*. A very important point is the occurrence of albuminuria in yellow fever. In *malaria* the spleen is enlarged and the parasite is found in the blood. In *Blackwater fever* there is a green vomit, which may cause it to be diagnosed as Yellow fever, but it is accompanied by hæmoglobinuria, and no blood corpuscles are found in the urine; whereas in Yellow fever, if the urine is red, it will be found that the condition is due to the presence of blood corpuscles. *Acute Yellow Atrophy* of the liver has a more gradual onset, and is practically confined to the female sex.

*Etiology.* Yellow fever is peculiar to the West Indies, certain parts of America, the Brazilian ports, and the west coast of Africa. It is found only in seaport towns. It rapidly spreads, especially in those parts which are crowded and dirty. It is always worse in the summer months, as a high temperature is necessary for the existence of the poison. A slight frost will destroy it, as at Memphis in 1879. Formerly it was not thought to be directly communicable from man to man, and some still hold that view. Murchison believed it to be contagious. It certainly spreads through the air and by fomites. It was brought to this country by a ship in 1865. Clinically it resembles an intense form of *malaria* (jungle fever), and possibly like that it may be conveyed by a mosquito. It is usually stated that negroes do not contract the disease; but I am informed on good authority that this is not correct.<sup>1</sup> The disease does not spread so rapidly amongst them as amongst Europeans, probably because many of them have been rendered immune by a previous attack.

*Prognosis.* The case-mortality varies in different epidemics from 5 to 75 per cent. Sometimes the patient recovers uninterruptedly after the fever falls on the 4th day; in such cases the skin is moist, there is little albumen or vomiting, and little or no yellowness. On the other hand, death may occur with "typhoid state" a few hours after the onset of disease, or from collapse after the fever remits. The prognosis is always grave when the jaundice is intense, the vomiting frequent, and hamorrhages occur from the stomach or elsewhere.

*Treatment.* As the liver is found after death to be in a state of fatty degeneration, one of the first indications is to diminish the work of the portal system by attention to the dieting of the patient. Only weak milk and water, or mutton broth may be given. No

<sup>1</sup> Dr. F. B. Archer, Quarantine Health Officer, Barbadoes.

solid food must be taken for four days. Other treatment is symptomatic; *e.g.*, ice and astringents for the hæmorrhage. It is important to induce free elimination by the skin and kidneys. The rest of the treatment is conducted on the same principles as typhus fever.

§ 374. **Epidemic Cerebro-spinal Meningitis**<sup>1</sup> is characterised by:—(1) Fever, sometimes very irregular at the onset, becoming normal for a day or two, then rising again. It may be remittent, but not often. It is rarely over 102° to 104°, but may be considerably raised towards the end. The pulse frequency is not always relative to the degree of fever. (2) Symptoms of irritative intracranial inflammation such as very severe headache, of sudden onset, with vomiting and muscular spasm. Compression symptoms may supervene later. The so-called "Kernig's sign" is said to be a diagnostic point, *i.e.*, when the thigh is flexed on the abdomen the leg cannot be extended because of spasm of the flexors of the thigh.<sup>2</sup> (3) There is always retraction of the head, and sometimes opisthotonos may be present owing to the rigidity of the muscles of the back. Hyperæsthesia, especially along the spine, and severe pain in the back, may be so great that all movement is intolerable. (4) A prominent feature is the presence of some skin affection, very often occurring symmetrically. Herpes labialis or zoster are usually present. On the second day a rash of purpuric spots commonly appears on the neck and extensor aspects of the limbs. Urticaria and erythema may occur.

**Diagnosis.** This disease has to be diagnosed from *tubercular meningitis*, which has an insidious onset, and no eruption. From other forms of *meningitis* it is known by their having no eruption. When an epidemic is present there is little difficulty in the diagnosis. Cerebro-spinal meningitis may occasionally complicate pneumonia.

**Etiology.** The disease attacks persons under 20 usually, and males more than females. It never occurs in summer, and is most frequent in winter and spring. It does not appear to be contagious, although it usually occurs in epidemic form. It is probably of microbic origin, and the organism is closely related, if not identical with the *diplococcus* of pneumonia.

**Prognosis.** The disease has a case-mortality of 30 to 70 per cent. The usual course of the malady is three weeks; but there are three varieties based upon the duration besides the common form above described:—(i.) the foudroyant form, which kills the patient in a few hours or days; (ii.) the typhoid form, which lasts for several weeks; and (iii.), the form which recovers in a few days. The prospect of recovery is not good when the disease attacks infants or old people. Amongst the unfavourable signs are the occurrence of hyperpyrexia, convulsions, irregular breathing, or an unduly prolonged period of illness. The more common complications are inflammation of the joints, optic neuritis, and polyuria. A trace of sugar may appear in the urine. Amongst the sequelæ we may mention deafness, impairment of the vision, chronic hydrocephalus, and transient paralysis of the limbs or aphasia.

**Treatment.** In addition to the treatment given in all intracranial inflammation (Chapter XIX.), quinine should be taken in large doses.

§ 375. **Relapsing or Famine Fever** [Syns. Recurrent or Relapsing Typhus; Spirillum fever (Vandyke Carter)] is a contagious fever met with in times of famine, ending abruptly on the 5th, 6th or 7th day, and followed after an interval of one week without fever, by a relapse similar to, but shorter than, the first attack. The incubation period varies from 5 to 9 or more days.

**Symptoms.** (1) The fever has a sudden onset and rises rapidly. It frequently reaches 108°, a range which in other diseases is not consistent with life. After remaining elevated for 6 or 7 days the temperature returns to normal as rapidly as it rose. The fall is preceded and attended by profuse perspiration or diarrhœa, or both. This is followed by an interval of about a week, during which the patient feels exhausted, and the pulse and temperature are subnormal. At the end of this week a relapse occurs which is similar to the first attack, but shorter, lasting 3 or 4 days. In rare cases there is a second and even a third relapse. (2) Abdominal pain and tenderness, and great enlargement of the spleen and liver are present in almost all cases. Jaundice is also very common. Epistaxis is common, and sometimes there is vomiting of blood. Delirium is very rare, but if present is of the noisy kind, and occurs at the crisis. Convalescence is slow. (3) The *Spirillum* is found in the blood during the pyrexial period, but in the intervals it is only present in the spleen.

**Diagnosis.** The diagnosis is not difficult, on account of the circumstances under which the disease occurs, and the course of the temperature. *Enteric fever* and *small-pox* have a rash; rheumatic fever has joint lesions. Yellow fever, which it most resembles, has jaundice, and a diagnosis is only made by the course of the fever and the presence of the *spirillum* in the blood in relapsing fever.

**Etiology.** Relapsing fever is due to a specific microbe, the *spirillum*. The disease arises under the conditions which attend a famine, and has been noticed to accompany most epidemics of Typhus; in which circumstances the epidemic begins with Relapsing Fever and ends with Typhus. The poison is given off by exhalations and the skin; the infecting distance is extremely short. The disease appears in seasons of unusual distress.

<sup>1</sup> The author is mainly indebted for this account to "The Etiology and Diagnosis of Cerebro-Spinal Fever" (The Cavendish Lecture, 1899), by Prof. W. Osler.

<sup>2</sup> Major Buchanan, *B. M. J.*, vol. ii., 1899, p. 1412; and Epitome, *B. M. J.*, Sept. 16, 1899.



as during strikes. It does not occur with ordinary destitution, but in times of famine, when people eat unwholesome articles such as grass, roots, hay, &c. Overcrowding is not absolutely necessary for its production, in which respect it differs from Typhus. One attack does not confer immunity from a second. As regards the *predisposing causes*, age has no influence, nor have seasons or occupation.

*Prognosis.* The case-mortality is not nearly so great as that of Typhus; it rarely exceeds 25 per cent. Age has not much influence, but dissipation and debility are unfavourable. Death, which occurs generally at the height of the first attack, is usually due to syncope, from hæmorrhage or from granular degeneration of the heart. When occurring later, it may be due to complications. Untoward symptoms are:—more than one relapse, hæmorrhage, suppression of urine, the typhoid state, cerebral symptoms, or indications of a weak heart. A rapid pulse, a high temperature, and even jaundice are not unfavourable.

Remedial treatment consists of the administration of salines and diuretics. At the commencement of an attack considerable relief may be given by an emetic or mild purgative. Digitalis may be required for the heart and chloral for the sleeplessness.

§ 376. **Thermic fever or Heat stroke** (Synon. Siriasis, Heat apoplexy, Heat asphyxia, Sunstroke, Coup de soleil), is one of the numerous varieties of tropical fevers about the pathology of which we know but little.

*Symptoms.* The onset is usually sudden, during or after exposure to a hot sun. In some cases there are a few days prodromata consisting of headache and malaise. Then a short stage of delirium rapidly sets in, and is immediately followed by coma and high fever (108-9°). During the stage of delirium the patient is restless, with muscular twitching and spasms. The stage of coma is marked by a very hot skin, rapid pulse, flushed face, heavy or stertorous breathing, and contracted pupils. In most cases death occurs a few minutes or hours after the onset of insensibility.

*Diagnosis.* The coma of *uræmia* and other toxic states is known by the absence of high fever. In the coma of cerebral hæmorrhage into the pons fever may occasionally be present, but it would not precede the onset of coma. The comatose form of *malaria* has the parasite in the blood, and an enlarged spleen, but the conditions are difficult to diagnose.

*Etiology.* All ages and sexes may suffer. It is predisposed to by intemperance, fatigue, malaria, overcrowding, and weakness of any kind. The disease appears to be endemic in certain places, and at times an epidemic occurs. Samboni pronounces it to be due to a germ which requires for its action a high temperature. This would explain its frequent occurrence amongst those who have to perform long marches in the sun of tropical or sub-tropical climates.

*Prognosis.* The case-mortality is about one in four. Most patients die from failure of respiration after the onset of coma. Favourable cases terminate by crisis, and make a rapid convalescence. Much depends on prompt treatment.

*Treatment.* The indication is to reduce the temperature at once, if possible without the use of drugs. Lest ague be also present, it is best in malarial countries to give a hypodermic of quinine (gr. vii. every 4 hours) at once. In order to reduce the temperature the patient must be laid on a stretcher, with a sheet covered with ice placed over him. Iced water should be run over him till the thermometer in the rectum falls to 102°; or if much hyperpyrexia were present, to 104°. Then he should be wrapped in blankets, and stimulants given. Avoid strychnine because of the tendency to convulsions.

§ 377. **Hay Fever** (Hay Asthma), especially the constitutional variety, **Dysentery**, and **Cholera** give rise to a certain amount of pyrexia of a continued type.

HAY FEVER (§ 132) is recognised by the violent attacks of sneezing.

DYSENTERY (§ 221). Acute dysentery is sometimes attended at the onset by some degree of pyrexia, but much the most important symptom of this disease is diarrhœa.

In CHOLERA (§ 222) the abdominal cramps, collapse, and diarrhœa are the leading symptoms. During the collapse stage the temperature may be as high as 105° in the rectum, although in the axilla and mouth it is sub-normal. In the reaction stage, if the patient lives, there is usually a degree or so of pyrexia, lasting from a week to a fortnight.

Finally, there are several diseases which in their typical forms belong to Group III. or belonging to Group I. are seen perhaps before or after the eruption comes out, which may present pyrexia of a continued type. It is well in all cases of difficulty or doubt to remember this, and to pass in review the members of all three groups.

<sup>1</sup> *Lancet*, 1899, vol. ii., p. 609. Discussion at the Meeting of the British Medical Association.

## GROUP III. INTERMITTING PYREXIA.

§ 378. In this group of diseases the pyrexia is of an INTERMITTENT (or remittent) type, *i.e.*, the temperature drops at regular or irregular intervals to normal (or nearly to normal). This group is distinguished from Group I. by the complete absence of eruption. It is distinguished from Group II. mainly by the wide variations of the temperature.

<i>Common.</i>		<i>Rare.</i>	
I. Ague and other		VI. Enteric fever (some	
Malarial Fevers .	§ 379	cases) and occa-	
II. Latent Tuberculosis .	§ 382	sionally Influenza	§ 386
III. Visceral Syphilis .	§ 383	VII. Malignant Endocar-	
IV. Acute Septicæmia .	§ 384	ditis . . . . .	§ 386
V. Sub-acute Septic		VIII. Lymphadenoma .	§ 386
conditions . . .	§ 385	IX. Pernicious anæmia .	§ 386
		X. Opium habit . . .	§ 386
		XI. Multiple Sarcoma .	§ 387

The clinical investigation of these diseases is often attended by considerable difficulty. AGUE, which may be regarded as the type of this group, is essentially a *paroxysmal pyrexia*, each paroxysm having three stages (cold, hot, and sweating), and each paroxysm being usually separated by one or more days' interval of health. TUBERCULOSIS and SYPHILIS have a daily rise and fall, and are good examples of *regular diurnally* intermitting pyrexia. ACUTE SEPTICÆMIA, on the other hand, is noted for the *irregular* character and wide range of its temperature and the severity of the rigors. CHRONIC SEPTIC CONDITIONS occupy a position midway between these two types—regular and irregular intermitting pyrexia. In a given case of intermitting pyrexia which has arisen in a tropical and subtropical climate, ague is probable; but in England the commonest cause is probably latent tubercle. The MICROBIC REACTIONS aid us to some extent in the diagnosis of this group.

Turning to the rarer diseases, which must always be kept in mind. MALIGNANT ENDOCARDITIS is chiefly remarkable for the *long course* it may run. In LYMPHADENOMA we have the swollen *glands* to aid us; and in PERNICIOUS ANÆMIA the patient is a male and the skin is very sallow. MULTIPLE INFECTIVE SARCOMA is so rare as to be a clinical curiosity.

It follows therefore that if we have a patient's temperature chart before us, and it shows definite intermissions or remissions, the disease will belong to one of 3 sub-groups:—

A. REGULAR INTERMITTENT PYREXIA with 1 or 2 days INTERVAL, which CONTAINS only one disease—Malaria . . . . . § 379

B. REGULAR INTERMITTENT PYREXIA occurring DAILY, such as Tuberculosis, and Visceral Syphilis . . . . . §§ 382 *et seq.*

C. IRREGULAR INTERMITTENT PYREXIA, such as Septicæmia, and other pyogenic processes . . . . . §§ 384 *et seq.*

§ 379. **Ague.** (Syns. : Malarial Fever, Intermittent Fever, Remittent Fever, Jungle Fever.) Ague is a non-contagious fever due to the malarial parasite, occurring in paroxysms with complete intermissions.

*Symptoms.* As a rule the fever comes on suddenly without warning.<sup>1</sup> Ague has three characteristic stages. First there is a *cold stage*, in which the patient shivers or has a rigor, and feels cold, though the temperature is elevated 3 or more degrees; the skin looks cold, sometimes livid, and the nails are blue. This stage lasts from a  $\frac{1}{2}$  to 2 hours or so, and is followed by the *hot stage*, in which the temperature goes up to 103°—106°. It begins with flushing of the face, and is attended by headache, pains in the back and elsewhere. This stage lasts 3 or 4 hours, and is followed by the *sweating stage*, in which the perspiration is so profuse that the bed linen may be soaked. This stage lasts 1 or 2 hours and is accompanied by a fall of temperature. The spleen enlarges during the attack. The stages may be shorter or longer. The sweating stage is followed by an interval, during which the temperature is normal, or subnormal, and the patient is fairly well, except for great lassitude and indigestion. In the commonest type of the disease (tertian ague, Fig. 101), there is an interval of about 24 hours between the paroxysms, which if untreated may recur for weeks.

*Varieties of Ague.* Malarial fever may vary in two ways:—(a) according to the duration of the interval between the attacks; or (b) according to the intensity, both of which probably depend on the species of the plasmodium. (a) There are three well-marked types of periodicity (Fig. 101):—(i.) *Quotidian* ague, in which the paroxysm occurs daily, at about the same time each day; (ii.) *Tertian* ague, in which attacks occur every other day; and (iii.) *Quartan* ague, in which the attack occurs every third day. Various compounds of these occur. (b) The chief of the more severe forms are:—

<sup>1</sup> The period of incubation varies considerably. Sometimes there may be none at all; sometimes it is not more than a few hours; while in some cases there may be an interval of months or years after exposure before the disease develops. Dr. Patrick Manson gives 2 to 2½ years as the limit for tertian ague, and 2 to 3 years for quartan.

<sup>2</sup> Here, as in all the diseases of this group, the elevation of temperature may be overlooked owing to the apyrexial intervals, and then we have only the patient's statement that he has had "chills" and perhaps sweatings to guide us. On the other hand we must be careful not to be led astray in this way. For instance, one patient came to me with this story, who had no fever at all; the symptoms of flushing and sweating had been induced by alcohol administered for shivering due to a neurotic condition.

(i.) The *comatose*, in which the patient suddenly passes into coma; (ii.) the *hyperpyrexial*, in which the temperature rises suddenly to  $107^{\circ}$  or  $112^{\circ}$ , and death occurs in a few hours; (iii.) the *algid*, which resembles the algid stage of cholera. Other varieties depending upon the intensity are referred to below, namely, Remittent or Jungle Fever, Algid, Comatose and Hæmorrhagic Malaria, and Blackwater Fever (!). *Æstivo-autumnal* fever is a

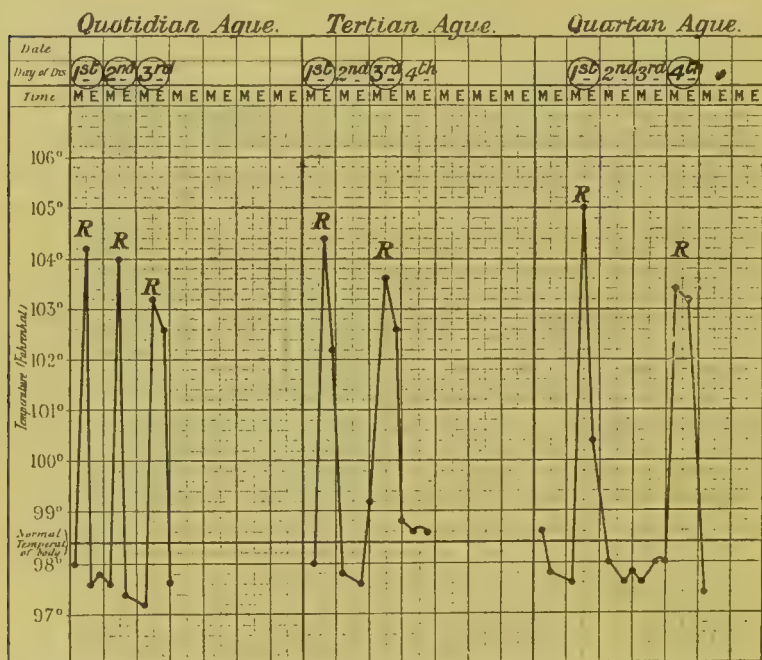


Fig. 101.—TYPES OF AGUE.—Quotidian (daily); Tertian (every other day); and Quartan (every third day). "R" indicates the rigor which ushers in the cold stage.

variety of malarial fever attacking Europeans on the Gold Coast and elsewhere. It is conveyed to man by various kinds of mosquito (*e.g.*, *anopheles pictus*, and *a. costalis*), and is also connected, by Dr. A. J. Chalmers, with the *anopheles Kumasii*.<sup>1</sup>

*Diagnosis.* Malaria is not easily, and is in point of fact rarely mistaken for other diseases; but the other disorders attended by intermitting pyrexia about to be described are very frequently mistaken for ague. *Clinically*, this mistake would be avoided if

<sup>1</sup> *The Lancet*, Nov. 3, 1900, p. 1262.



it were remembered that *ague of quotidian periodicity*—daily recurrence—is very rare;<sup>1</sup> and that tertian or quartan periodicity is absolutely pathognomonic, it occurs in no other disease. In leprosy and all the diseases mentioned below, the intermission is daily. *Therapeutically*, the diagnosis may be established by full doses of quinine; if this be hypodermically given and fail to relieve, the attacks are certainly not malarial. The *microscopic* diagnosis of the parasite in the blood requires considerable experience, but it is always possible to find it in blood-films, provided the patient has not taken quinine for several days; this, of course, is positive evidence.<sup>2</sup> Enteric fever and many other conditions belonging to Group II., when occurring in a malarial subject, are apt to assume a malarial or intermitting type of pyrexia.

*Etiology.* Age and sex have no real influence. The disease is most prevalent at the latter part of the rainy season. Debility, intemperance, exposure to chill, and especially exposure to the night air,<sup>3</sup> are regarded as predisposing causes. One attack predisposes to a second one; indeed, when once a person has contracted malaria, he is always liable to it for many years (see footnote, p. 635). The *exciting* cause of malaria is a parasite, the plasmodium (see Bacteriology) introduced into the blood, the nature of which has only been discovered within the last few years.<sup>4</sup> It is introduced into the blood of the patient by the bite of the Anopheles, a mosquito, which serves as an intermediate host for the parasite. The disease is endemic in certain districts, which are called malarial, and these districts are always situated in tracts of country which are marshy, or where the soil is moist and covered with pools of water and decomposing vegetable matter. It is still on rare occasions said to be met with in some parts of England, *e.g.*, Isle of Thanet, Cambridgeshire, and Lincolnshire;

<sup>1</sup> The only fallacy to this statement occurs in those rare cases when both the tertian and the quartan parasites affect the patient at the same time and thus give a partial semblance to quotidian attacks. But even then the pyrexial attacks are not really quotidian, as may be seen from the following diagram. Supposing "a" represent the attacks of a tertian parasite, and "b" those of a quartan parasite, there would be one day's interval after at longest three days pyrexia, viz. :—

a	b	a	b	a	b	a	b
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<sup>2</sup> Dr. Patrick Manson, *The Lancet*, May 17, 1902.

<sup>3</sup> Now known to be due to the fact that mosquitoes are most active during the night.

<sup>4</sup> Reports of Koch's investigations into Malaria, *Brit. Med. Jour.*, vol. i., 1900, pp. 325, 1183, 1597.

but it is chiefly in the dense forests and uncultivated tracts in Africa, Asia, and South America that the disease is prevalent. It



Fig. 102.—Mosquitoes settling on a wall. The genus *Anopheles*. There are two chief types of mosquitoes—*Culex* and *Anopheles*—easily differentiated by their attitudes when resting upon a wall. *Anopheles* is the more dangerous one and is recognised by its spotted wings and its tilted attitude. Its larvæ lie flat on the surface of puddles, and move along the surface; whereas *Culex* larvæ lie more perpendicularly and if disturbed rush to the bottom of the pool. *Anopheles* larvæ are found in puddles which contain algae and which are too large to be dried up in a week (time needed for the mature insect to be hatched). They are not found in pools which contain minnows, nor in rapid streams, nor in shallow rain pools that are easily dried up. Kerosene oil (about 3i to a pool of 1 square yard) killed all larvæ in 6 hours. "Malaria Expedition to Sierra Leone," *Brit. Med. Journ.*, Sept. 30, 1899, p. 869. See also meeting of Path. Soc. at Cambridge, Summer, 1900; *Lancet*, vol. ii., 1900 (Nuttall); and Ross and Mallerd, *Brit. Med. Journ.*, Nov. 3, 1900.

disappears from a district when the soil is drained and cultivated. Standing water, especially in puddles, seems to be a necessary condition, together with a moderately high temperature. Malaria was always observed to keep close to the ground, and its spread was known to be interrupted by a tract of water, especially salt water, or by a grove of trees. It was known that change of wind would bring malaria from a distance. Persons newly arrived in a district were especially prone to contract the disease. These and other curious data have been known for many years but remained without explanation until, quite recently, it has been proved by Ross, Nuttall, and others, that the disease is conveyed, and is introduced into the blood of man, by certain varieties of mosquito (Fig. 102).

*Prognosis.*—Death usually occurs from complications, without which malaria is not a very fatal disease. The most favourable type of case is that in which the pyrexia runs a typically intermittent course. The gravest,

and happily the rarest, is that form in which the pyrexia is continued or only remittent. Coma or delirium, hæmorrhage from the stomach or bowels, and choleraic diarrhœa with cramps are

unfavourable signs, and if collapse sets in after the hot stage, a fatal termination is usual. The chief *complications* are (i.) great weakness and anæmia, which are common results of the disease; and in time, especially if untreated, the patient develops the typical cachexia of malaria. Pigmentation of the skin is a marked characteristic of this cachexia—a general distribution of pigment, but especially around the eyes.<sup>1</sup> This is doubtless accounted for by the deposit of blood-pigment granules which are so constantly found in the blood. (ii.) Enlarged spleen, “ague-cake” (§ 265, VI.), is a usual sequence, and rupture of the organ occasionally takes place. (iii.) Jaundice, due usually to hepatitis, is one of the more serious complications, and the liver after many attacks becomes enlarged.

*Treatment.* Quinine is a specific in the treatment of ague. The hydrochlorate or the sulphate, dissolved in tartaric acid and water is preferred to hard and sometimes insoluble pills and tabloids. A large dose (at least gr. x.) should be given, and repeated in diminishing doses four times daily. Some advise that it should not be given in the pyrexial stage. An aperient should have been previously administered. When prompt action is required (as in the graver forms), 7 to 10 grs. of the acid hydrochloride of quinine should be given hypodermically; 1 or 2 doses being sufficient. For the resulting anæmia arsenic with iron is especially useful, and Manson recommends that quinine with salines be taken once a day for two months. Stimulants are also called for in the severe varieties—ether, strychnine, or brandy per rectum. For “ague-cake,” iodide of mercury rubbed in over the enlarged organ is said, by foreign observers, to be of value. The indications for *prophylactic* treatment are based upon the etiology. Marshy tracts must be drained, and of all trees the red gum tree, from its rapid growth, is the most suitable for this purpose. The night air must be avoided, and high sites selected for sleeping. Quinine, 2 to 5 grs. 3 times a day, given as a matter of routine is certainly a preventative. It is now recommended that the larvæ of the anopheles mosquito should be killed by pouring kerosene oil upon the stagnant pools and puddles in the district (see Fig. 102).

<sup>1</sup> On one occasion when I joined a passenger ship from the East this periorbital pigmentation was so marked in one of the passengers that I thought he must have been fighting, until I learned that he had been a victim of malaria.

§ 380. **Remittent Fever** (Syn. : Jungle Fever, Continuous Malarial Fever). Remittent fever is now generally admitted to be a variety of Ague in which there is a protracted hot stage, and no apyrexial intermissions. The cold stage is either absent altogether or is only marked by a chilliness. The pyrexia continues, with slight daily remissions, for a week, two weeks or more. In some forms there is a tendency to the typhoid state ("typhoid remittent fever"). In other forms gastric symptoms are prominent, together with marked jaundice ("bilious remittent fever"). Sometimes there is great prostration, with hæmorrhages; and this form is followed by extreme anæmia. The *diagnosis* from Yellow Fever on the one hand, and Enteric on the other, may be impossible without an examination of the blood. In Yellow Fever albuminuria is present, and the temperature falls in 3 or 4 days. Enteric gives the Widal reaction. The *prognosis* is always unfavourable. It is especially bad when the remissions become less marked, and the typhoid state supervenes. Unfavourable symptoms are, collapse, delirium and coma.

§ 381. "**Blackwater**" **Fever** (Syn. Hæmoglobinuric fever), so named from the colour of the urine, is a non-contagious endemic fever. It is supposed by some to be really one form of malaria, but definite proof is wanting; others consider it is due to a special plasmodium.

*Symptoms.* In a typical attack the onset is marked by rigors, and the temperature ranges from 103° to 105°. The urine which is passed is of a dark colour, due to the presence of hæmoglobin; it is scanty in amount and of high specific gravity. There is bilious vomiting, which may be extremely severe, and accompanied by intense jaundice. As the fever falls the urine clears; then a new paroxysm of fever may set in with a return of the hæmoglobinuria. The liver and spleen may be enlarged. During the paroxysm there is great destruction of red blood corpuscles, and the blood shows poikilocytosis. *Etiology.* This fever is endemic in the tropical and subtropical regions of America and Africa, especially West Africa. It attacks those in particular who have been rendered weak by attacks of malaria.

*Diagnosis.* This disease may be mistaken for Yellow Fever, but in the latter one attack renders immunity, whereas in Hæmoglobinuric Fever there is usually a history of many attacks. In Yellow Fever the spleen is not enlarged, and the fever does not remit several times in succession.

*Prognosis.* The case-mortality is 25 per cent. Frequent relapses are certain to occur if the patient remains in the endemic district; even if he return home he is liable to have attacks of hæmoglobinuria, though these may be accompanied by little or no fever. In severe cases there may be profound prostration, with all the symptoms which accompany a profuse hæmorrhage. Death may result in this way or from syncope, or collapse. There may be suppression of urine, and death with symptoms of uræmia.

*Treatment.* Manson advises the avoidance of quinine, unless the presence of the malaria parasite in the blood indicate its necessity. Otherwise the de hæmoglobinating effect of quinine will only do harm. The patient must be kept at rest, and water may be freely given. Iron is indicated on theoretical and practical grounds.

§ 382. **Latent Tuberculosis.** Tuberculosis is said to be latent when the usual physical signs or local manifestations are wanting. In all cases of unexplained intermitting pyrexia in this



country one of the first things to be suspected is tuberculosis in some part of the body. It may be very deeply seated but it is a useful clinical axiom to remember that *no active tuberculosis can exist in any part of the body without the occurrence of a daily intermitting pyrexia*. Moreover the degree of the fever is a fair indication of the activity of the process. The chart is a typical one, the temperature drops each morning to (about) normal and rises each evening 1, 2, or more, degrees; occasionally *vice versâ*. The physical signs may be altogether wanting and the patient perhaps only seeks advice on account of the weakness, dyspepsia, and other vague symptoms. Such a condition may go on for weeks without any local manifestations, as in the cases referred to under Tubercular Meningitis.<sup>1</sup> The lungs, kidneys, peritoneum, and various organs may be affected. (1) The commonest locality in adult life is the *lungs*. In this case physical signs usually appear which resemble bronchitis or simple pulmonary congestion, for which diseases it is apt to be mistaken (§ 87). (2) The *meninges*, *peritoneum*, and other *serous membranes*, are perhaps the commonest positions in childhood in which tubercle may be deposited without definite signs. (3) In the *kidney*, tubercular pyelitis may be readily overlooked; and in suspicious cases the urine should be carefully examined for traces of pus and tubercle bacilli (§ 306). (4) Tubercle may also be latent in other situations such as the cranium, spine, intestines, and other viscera; and finally, the tuberculous process may be generalised, and give rise to the condition known as *Acute General Tuberculosis*.

§ 382a. **Acute General Tuberculosis** (Syns. Acute Miliary Tuberculosis; Typhoid Tuberculosis) is an obscure disorder characterised by intermitting pyrexia, prostration and a tendency to the typhoid state—due to a generalised infection of the body by the tubercle bacilli.

*Symptoms.* (1) The onset is most insidious. The patient complains perhaps of nothing but lassitude, which is attended by feverishness of a typical intermitting type, and perhaps bronchial catarrh. The temperature each morning may be normal, that in the evening raised one or more degrees. The inverse type, *i.e.*, a lower temperature in the evening than the morning, is said by some to be more frequent in this than in other

<sup>1</sup> Some years back I saw a case several times with Dr. A. J. Hubbard, of Hemel Hempstead, occurring in a man aged 27, who had an elevation of temperature of a diurnally intermitting type, without any other symptoms excepting headache and prostration. Thorough investigation revealed no physical signs anywhere. The intermitting pyrexia made us suspect Tuberculosis, and the extreme severity of the headache suggested Tubercular Meningitis; but it was not until the end of 9 weeks that optic neuritis, delirium, and ocular paralysis supervened to confirm the diagnosis.

forms of tuberculosis.<sup>1</sup> In very rare cases the highest daily temperature does not rise above normal. The patient complains of lassitude, which gradually increases, and in the course of a few weeks he has wandering muttering delirium, at first only at night. Maniacal delirium is rare. The typhoid state supervenes towards the end. (2) The respiration is always increased in frequency. The pulmonary signs, which are generally present, have been mentioned (§ 87). (3) As a rule there are no marked local manifestations.

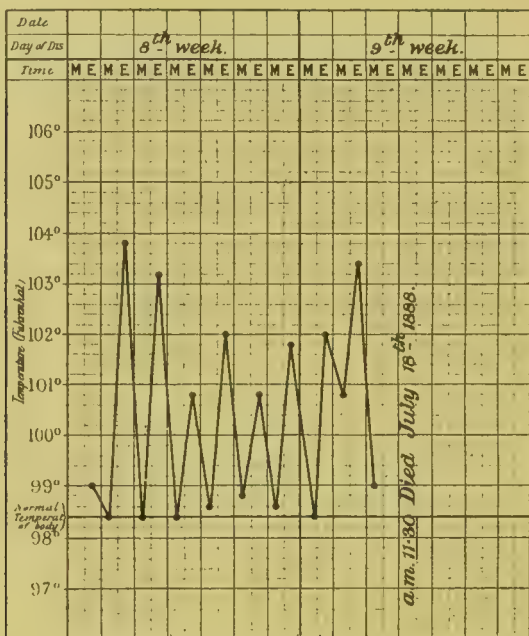


Fig. 103.—ACUTE MILIARY TUBERCULOSIS. Geo. W—, æt. 49, admitted to the Paddington Infirmary, July 9th, and died July 18th, 1888. 7 weeks' history of vague illness before admission, during which time there was profuse hæmoptysis on one occasion. The signs in the chest were very indefinite during life. After death the lungs were sparsely studded with miliary tuberculosis. The liver and peritoneum were also dotted with tiny tubercles, hardly visible to the naked eye.

but, according to the chief seat of mischief, various other signs may be elicited, such as paralysis of the cranial nerves, peritonitis, pleurisy. The spleen is nearly always enlarged.

*Diagnosis.* 1. The presence of bacilli in the sputum is pathognomonic, and these should be looked for repeatedly in all cases of "bronchitis" attended by an intermitting pyrexia, especially in young adults. Most cases of Acute Miliary Tuberculosis in the early stage are admitted to hospital as bronchitis, in the later stages enteric fever. 2. The course of the

<sup>1</sup> According to Reinhold (quoted by Osler, *loc. cit.*), 18 per cent. of tuberculosis cases present an inverse temperature.

disease may have so close a resemblance to *enteric fever* that Niemeyer (before the discovery of the tubercle bacillus) stated that this disorder might be indistinguishable until the patient reached the dead-house. The Erlich Diazo-reaction occurs in both Enteric and Acute Tuberculosis, but not the Widal reaction.

*Etiology.* The disease is due to a general dissemination of the tubercle bacilli throughout the body. This may have been introduced from outside, but far more frequently it can be traced to some chronic or subacute focus in the patient himself. This may be an old caseous or fibroid gland which appeared to be dead, or an old quiescent spot in the lungs or elsewhere (see footnote).

*Prognosis.* The disease is uniformly fatal in the course of 4 to 8 or more weeks. Death occurs by coma, sometimes by pulmonary or other complications. The height and range of the temperature is a fair measure of the virulence and activity of the morbid process.

*Treatment.* In such widespread mischief no treatment is of any avail. As regards prevention two important points should always be remembered ; (1) that convalescence from pulmonary tuberculosis should always be very thoroughly re-established, before treatment is stopped ; and (2) tuberculous glands should always be removed, even when they appear to have undergone spontaneous subsidence.<sup>1</sup>

§ 383. **Visceral Syphilis.** It is now generally recognised that syphilis is a specific contagious disease like small-pox. There are two different stages of syphilis at which intermitting pyrexia may occur. (a) At the first development of the primary roseolous eruption fever, there may be some elevation of temperature.<sup>2</sup> This is generally overlooked ; but at other times it may be accompanied by thirst, loss of appetite, shivering, etc. This pyrexia always occurs within 65 days of the date of the infection, and it is only present if no mercury be given. (b) In the later secondary and tertiary stages of the disease an intermitting pyrexia may occur in connection with syphilitic periostitis, or gummata of the internal organs.<sup>3</sup> This is a not infrequent occurrence in the course of clinical work, and syphilitic lesions of this kind are always to be suspected in cases of prolonged

<sup>1</sup> I remember a case of a young woman who came under my care for paralysis of one arm due to the pressure of tuberculosis of the glands on the nerve trunks in the posterior triangle. Under constitutional treatment the glands subsided and she completely recovered the use of her arm, but refused to have an operation for the removal of the glands. Three years later she died of acute military tuberculosis.

<sup>2</sup> This has only been known of late years. It was first pointed out by Guntz in 1865, and called general syphilitic fever. Lancereaux also pointed it out in 1866, and stated that it much resembled quotidian ague. He referred to several cases.

<sup>3</sup> Two cases are reported by Dr. Alfred Dufferin in the Clin. Soc. Trans. 1869. The following year a Committee reported 9 cases. Dr. Bristowe reports a case of gumma of the liver in a lad of 16, in whom the temperature went up every evening 2° or 3°, the cause being overlooked until interstitial keratitis was discovered, and iodide was given (Clin. Soc. Trans., vol. xix.) The author has records of 6 similar cases in which the leading symptoms were intermitting pyrexia, anæmia, and signs referable to the liver or spleen, all of which rapidly disappeared under iodide—see also Clin. Journ. Dec. 1, 1897, p. 87.

intermitting pyrexia, especially if it be attended by anæmia. The morning temperature is normal, but in the evening it goes up 1, 2, or more degrees (Fig. 104). There may also be rigors, nocturnal sweating, and paroxysms of pain in the joints, unrelieved until iodides are given; then the symptoms speedily subside. In obscure cases careful investigation should be made of the eyes, liver, ribs, clavicles and other bones.

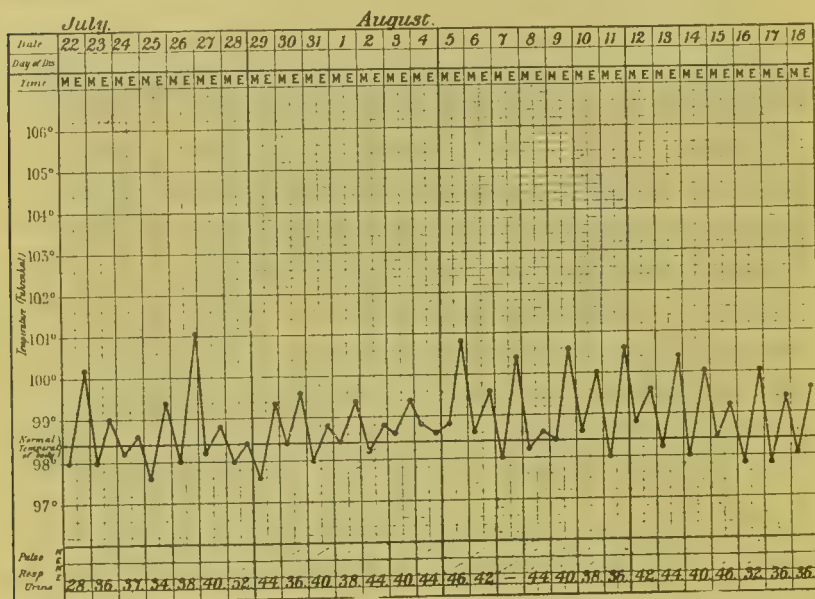


Fig. 104.—VISCERAL SYPHILIS. Annie L.—, æt. 66, admitted to the Paddington Infirmary, July 22nd, 1889(?). The temperature subsided under iodide in large doses, but she ultimately died of exhaustion and hypostatic pneumonia. P.-M.—Gummata of liver and bones, hypertrophic cirrhosis, widespread fibrosis of organs.

§ 384. **Acute Pyæmia or Septicæmia**<sup>1</sup> (Syn. *Ichoræmia*) is a disease characterised by a wide range of temperature, accompanied by rigors and sweating, due to the direct introduction into the blood or lymph—usually through some breach of surface in skin or mucous membrane—of a pyogenic microbe, of which there are several.<sup>2</sup>

The *symptoms* are :—(1) Pyrexia, which runs a very characteristic course, and is distinguished from all other diseases not of septic origin by the *wide* and *very irregular* range of the temperature

<sup>1</sup> There is still some confusion in the use of these terms, but for clinical purposes they may be regarded as synonymous. In former times, when localised deposits of pus occurred the former term was generally applied, when these were absent, the latter.

<sup>2</sup> As to the nature of these organisms compare footnote, p. 647 and p. 661.



(Fig. 105). The remissions may occur several times a day, and have not the diurnal regularity which marks the two preceding

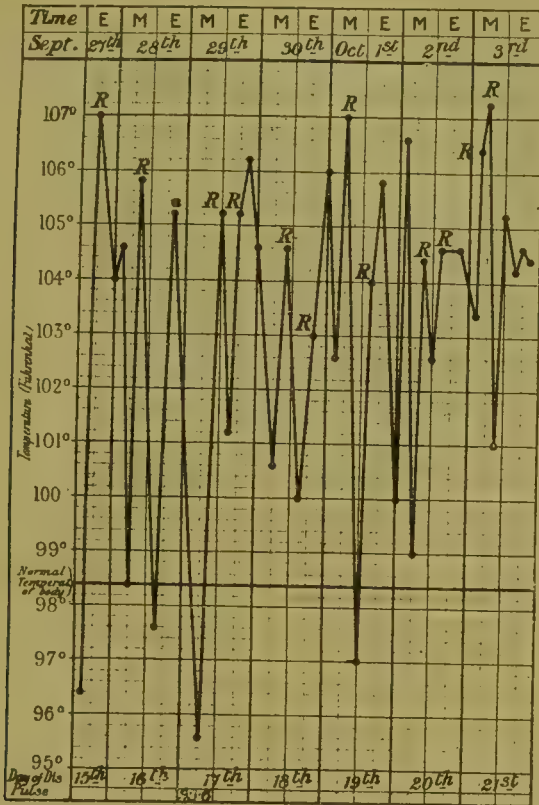


Fig. 105. -ACUTE SEPTICÆMIA (typical of an irregularly intermitting pyrexia). Catherine W—, æt. 6, admitted to Hospital, September 27th, 1881. She was taken ill somewhat suddenly on September 13th with shivering and vomiting. On admission she was in a condition of prostration. There were no physical signs excepting a systolic bruit over the whole cardiac area, and slight enlargement of the spleen. On the 30th there was rusty sputum with streaks of blood; dulness and crepitations over the right back. There was delirium from time to time, and she died somewhat suddenly on October 3rd. At the autopsy there was found pus in the mastoid cells and sinus thrombosis secondary to long-standing middle ear disease, of which a history was now obtained, infarcts in the kidney, and pyo-pneumothorax secondary to rupture, of one of the gangrenous-looking abscesses of the lung.

classes of disease (§§ 382 and 383). There may be as much as 6 or 7 degrees difference between the temperature in the course of a few hours. When at its highest point the temperature is accompanied by a rigor, followed by very profuse perspiration and

a rapid fall. The pulse is rapid and compressible, and the prostration and lassitude are very marked. The mind is clear at first and remains so for a considerable time, but towards the end there is a tendency to the typhoid state. (2) Nausea, vomiting, and diarrhœa are common, the skin is sallow, and there is often jaundice. (3) Later on in the disease emboli and deposits of pus (pyæmic abscesses) may occur in different parts of the body, especially in the lungs, giving rise to a generalised congestion and patches of pneumonic consolidation or gangrene (as in the case given in Fig. 105), in the liver and spleen; and deposits of pus may occur in or around the joints. The serous cavities may also contain pus, and we get empyema and pyo-pericarditis. The occurrence of albumosuria is an indication of a focus of pus in the body, and this may be an aid to diagnosis; so also are the leucocytosis and other changes in the blood (Chapter XX.).

*Acute Osteomyelitis* (or, as it used to be called, Acute Periostitis or Acute Necrosis) is a pyæmic process which may set in very suddenly, usually after an injury to one of the superficial bones, generally the tibia. In children there may be no history of injury. The diagnosis is easy when the tissues round the diseased bone are swollen; but during the first day or two of the disease pain is often complained of near a joint, and may lead one to diagnose Rheumatic fever.

The *diagnosis* of septicæmia is easy when there is an external wound or abrasion, and should never be difficult on account of the wide variation of the temperature, coupled with the rigors and the sweats. The chart of a typical acute case is like nothing else. When due to some internal cause it may resemble malignant endocarditis, enteric fever, pneumonia, ague, remittent fever, and acute rheumatism. But when carefully recorded temperatures of several days are available, and a thorough examination of the organs is made, the diagnosis should not be difficult.

*Etiology.* A cause—*external* or *internal*—for pyæmia should always be carefully sought. Among *external* sources, unhealthy wounds were, before the introduction of Listerism, a prolific source of this disease, and the patients in the surgical wards of olden times were decimated by it. A mere scratch is sometimes sufficient for the introduction of the poison, and sometimes the most trivial operations are followed by pyæmia. The source of infection may arise from some *internal* condition, and in these circumstances

the pyæmia used to be called "idiopathic." The internal sources are very numerous—sometimes it is caries, especially of the mastoid bone, sometimes periostitis, or osteomyelitis, sometimes an ulcer and other breach of surface in the mucous membranes. Ulceration of the biliary passages, and of the urinary passages are frequent sources of infection. Special attention should be directed to the vermiform appendix (see Appendicitis, § 176) and the uterus. *Recent abortion, perhaps criminally procured, should always be borne in mind when a young woman is admitted with septicæmia.* After recent parturition, the uterus resembles an open wound and offers a large surface for the absorption of the pyogenic poison, hence the frequency with which septicæmia complicates parturition unless the most scrupulous cleanliness has been observed. The disease is then called Puerperal Fever or PUERPERAL SEPTICÆMIA. When the poison is derived from a previous case of puerperal septicæmia it is specially virulent and fatal.<sup>1</sup> Among the *predisposing causes*, overcrowding, bad ventilation, want of cleanliness, and other unhygienic and septic conditions, are among the most fruitful.

*Prognosis.* The course of septicæmia differs widely. Thus, on the one hand, some cases of intense septic intoxication, due for instance to infection from a *wound* or parturition, run a rapid and fatal course of 10 to 12 days, terminating in the "typhoid state." On the other hand, cases in which apparently small quantities of septic matter are constantly leaking into the general circulation from some *internal* source may be indefinitely prolonged over many weeks or months, the mind remaining clear the whole time. Such would appear to have been the course of the disease in the patient referred to in Fig. 106. There is, in point of fact, no definite line to be drawn between the *acute* septicæmia now under consideration, and the *subacute* and *chronic* septicæmia due to pent-up pus or ulceration described below (§ 385). Acute pyæmia is a most serious, and if untreated, invariably fatal malady. Death may occur either by the intensity of the poison

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<sup>1</sup> When a puerperal case becomes infected with scarlatina or one of the other specific fevers, the disease is very virulent. The symptoms resemble those of septicæmia more than those of an infectious fever. These and other clinical and laboratory data lead to the belief that there are many different pathogenic organisms which may lead to the septic intoxication we know as Septicæmia (see § 380).

(typhoid state), asthenia, or complications. The *untoward symptoms* are, a very high temperature, frequent rigors, or cerebral symptoms. The most frequent *complications* are—(1) pneumonia, which invariably occurs in severe cases ; (2) pericarditis or pleurisy, which usually become purulent, and peritonitis ; and (3) suppurative inflammation of the spleen, liver, and other organs, consequent on the infective emboli. Among the sequelæ in certain less acute cases which recover may be mentioned a destructive form of arthritis.

*Treatment.* The indications are, (1) to remove the cause, (2) to inhibit the microbic toxin, (3) to relieve the symptoms and maintain the strength. 1. If the infection is derived from a wound or some accessible purulent cavity (*e.g.*, an abscess, an empyema, acute necrosis, etc.) this should be promptly laid open, drained, and treated by antiseptic measures. Search must be made for some internal cause (*e.g.*, appendicitis) and this should, if possible, be dealt with. 2. Thanks to the researches of modern pathology we are now in possession of a reliable serum for some cases, the anti-streptococcic serum ; and, at the time of writing, several cases are on record which have been rescued from death by this means.<sup>1</sup> As previously mentioned (and see § 389), several different bacteria may produce the disease, and we must identify which is in operation before we can employ the appropriate antitoxin. The most frequent, when the source is some purulent focus or abscess, is the streptococcus pyogenes. 3. The administration of quinine in large doses has some controlling influence over the temperature ; antipyrin, antifebrin, and other febrifuges are also used. The internal administration of antiseptics generally has not been found of much use. Stimulants and concentrated nourishment are called for (see also § 392 *et seq.*).

§ 385. **Subacute and chronic septic conditions** (*e.g.*, **abscess, ulceration, etc.**) also give rise to intermitting pyrexia. The various clinical conditions met with under this heading are due to the absorption of some septic or toxic material into the circulation. The possible sources of the sepsis are very numerous and may be grouped into two divisions—a, **ABSCCESS** (or pent-up

<sup>1</sup> Low, *Lancet*, 1898, vol. i., p. 779 ; Mitchell Bruce, *Lancet*, 1898, vol. ii., p. 515 ; Leask, *Brit. Med. Journ.*, June 20, 1896, p. 1500 ; Boune, in *Therapeutische Monatshefte*, Sept., 1898 ; and others.



pus), and b, **ULCERATION** (internal or external): clinically the former is more acute than the latter, and indeed the former might be called subacute, the latter, chronic septicæmia.

a. **ABSCCESS: PENT-UP PUS.** Pus never forms in any part of the body—*e.g.*, in the pleura (empyema), in the liver (hepatic abscess), or elsewhere—without the occurrence of “chills,” “shivers,” or “rigors,” and an intermitting or remitting pyrexia. Before the clinical thermometer was invented, these shiverings (sometimes followed by sweating) were the chief symptoms by which the formation of matter was identified. When there is fluid in the chest, for instance, and we do not know whether it is serous or purulent, the occurrence of shivering or sweating will often settle the question in favour of pus. The temperature in such cases presents much the same chart as that in tuberculosis, though it has not such regularly diurnal variations, and is more often accompanied by shivering or rigors. There are considerable lassitude, debility, pallor (though with a hectic flush on the cheeks), and more or less loss of flesh in course of time. Albumosuria is usually present, and is a valuable confirmatory symptom. Blood-films should always be examined, and the presence of leucocytosis with an increase in the proportion of polynuclear cells will afford strong confirmation that a pyogenic process is in operation.

*Causes.* Abscess or pent-up pus in any position may produce these symptoms; and careful search should be made for abscess of the liver, spleen, or other organs, pelvic cellulitis, caries of the spine or mastoid bone, appendicitis (Fig. 106), intracranial abscess, empyæma, pyo-nephrosis, etc. Pain is the chief localising symptom, but it may be wanting. On giving free exit to the pus the pyrexia should immediately subside.

b. **ULCERATION** of an **INTERNAL** or **EXTERNAL** surface (including the conditions known as “Hectic Fever,” Hepatic, and Urinary Intermitting Pyrexia), is always attended by some degree of intermitting pyrexia, running a more chronic course than the foregoing. This fever also differs from the last in the usual absence of definite rigors. Sometimes the shivering may not amount to more than “chills down the spine”—thought to be ague, perhaps—and sweating which is hardly noticed. The morning

temperature is normal, or almost normal, and it is raised one or two degrees some time during the day. Anæmia and failing health are always present; and here again albumosuria and the blood changes just mentioned are present. This kind of fever, due to prolonged suppuration, and attended by chronic wasting, was formerly known (and is still among surgeons) as *Hectic Fever* (ΕΚΤΙΚΟΣ, Gr. habitual). When due to a discharging sinus—a sinus, for instance, connected with caries, or necrosis of a bone, or a bed-sore—the cause is obvious. But the condition may also be set up

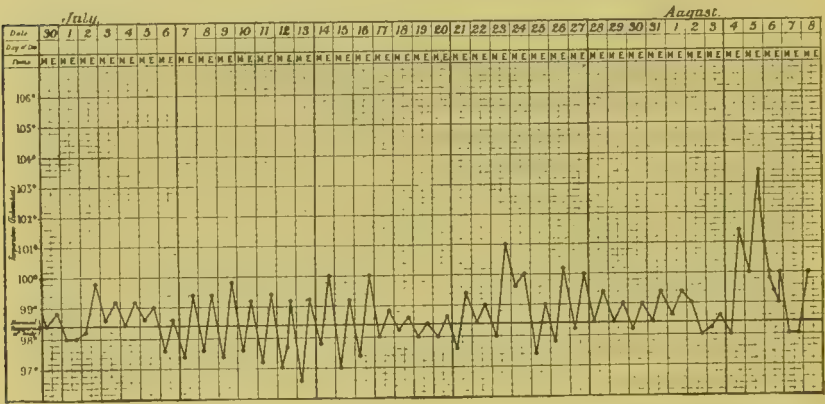


Fig. 106. —CHRONIC PYÆMIA. Frank T—, æt. 31, had had an attack of gonorrheal rheumatism two years before from which he had recovered. The present illness had come on quite gradually a month or so before admission. Stiffness and pain in the joints being the chief symptoms, and the urethra being *absolutely normal*, it was regarded as a case of chronic rheumatism, though none of the usual remedies had any effect. The joints became progressively worse, and though he complained of abdominal pains from time to time, attention was not directed to that cavity. He died some two months later suddenly from perforation of the appendix vermiformis. A review of the case pointed to a chronic septic process having its origin in the appendix, and specially affecting joints which had been previously diseased.

by ulceration of the intestines or any of the mucous membranes or internal passages (*e.g.*, the appendix, Fig. 106). It is called *Urinary Fever*<sup>1</sup> when it arises from chronic ulceration of some part of the urinary passages, *e.g.*, when a stone is impacted in the ureter, or when the patient has “stricture urethræ,” or there is ulceration of the pelvis of the kidney (pyelitis). This cause may be suspected if there be a history of renal colic. Similarly *Hepatic Intermittent Pyrexia* (ulceration of the biliary passages) may be suspected if there be a history of biliary colic. When the ulceration,

<sup>1</sup> The term *Urinary Intermittent Pyrexia* is applied by some to the rigors, sometimes accompanied by fever and sweating, which follow the passage of a catheter.

due to gall-stones, is situated in the *gall-bladder*, both colic and jaundice may be entirely absent, and the patient complains of nothing but the "chills" (§ 243).

§ 386. The **rarer causes of Intermitting-Pyrexia** are, with one exception (Multiple Sarcoma), fully described elsewhere, and need only to be mentioned here.

**Influenza, Enteric**, and other diseases described in Groups I. and II. are occasionally attended by pyrexia of an intermitting type. This is particularly so if the patient has had Ague or lived in a malarial district.

**Enteric fever** during the first two weeks of its course is attended by typically continued pyrexia; but in the concluding stage of the disease the pyrexia gradually drops each morning to normal, and the case may be seen for the first time in this stage. Under certain other circumstances also the temperature may be intermitting, viz., (i.) in rare instances it may commence with symptoms of Ague (Murchison); (ii.) in very mild cases the temperature may be intermittent; (iii.) after lasting a few days the fever sometimes aborts and takes on an intermitting type. For the diagnosis of the disease we now have an infallible guide in Widal's test.

Various **local inflammatory diseases**, other than the septic conditions previously mentioned, may at times be attended by intermittent pyrexia. In cirrhosis of the liver, for instance, a prolonged fever with daily oscillations has occasionally been observed.<sup>1</sup>

**Malignant Endocarditis** (multiple systemic embolism) (§ 42) is always attended by pyrexia of an irregularly intermitting type, sometimes with sweatings and rigors, very much resembling the chart of septicæmia, though the temperature is usually a little more diurnally regular, and rigors are not usually so frequent (compare charts Figs. 105, 107, and 15). The diagnosis of these two diseases is sometimes very difficult (p. 76). Malignant Endocarditis is favoured by (i.) the existence of a loud cardiac murmur detected quite early in the case; (ii.) a history of acute rheumatism. (iii.) The secondary emboli in this disease are more frequently found in the systemic arteries, such as those of the spleen, liver, and kidneys, and they do not result in abscesses. In pyæmia the emboli occur primarily in arteries of the lungs, and from the very beginning they suppurate and form abscesses which constitute centres of secondary infection elsewhere.

**Lymphadenoma** is recognised by the periodical enlargement of the lymphatic glands. This enlargement is attended by pyrexia of an intermitting character.

In **Pernicious Anæmia** the temperature is sometimes subnormal, but it is more frequently attended by exacerbations of fever of an intermitting type. Rigors and sweats may also occur, but they are not usual. The disease is also identified by the intense sallowness of the skin, and the fact that the patient is of the male sex, and advanced in life.

<sup>1</sup> Dr. Frederick Taylor, Clinical Lects. on Polyarteritis, *Brit. Med. Journ.*, Dec. 16, 1900.

The **Opium or Morphia Habit** is attended from time to time by attacks of intermittent pyrexia, during the reaction stage, in which there are cold, hot and sweating stages. Dr. Livenstein calls attention to this fact, and records cases where no other cause could be found, and where the attack ceased on giving opium.

§ 387. **Multiple or Infective Sarcoma** (Generalised Malignant disease). Neither sarcoma nor carcinoma is usually attended by an elevation of temperature. But in certain circumstances the disease seems to take on an infective type and spreads rapidly to other parts of the body, especially when the skin is primarily affected. Hilton Fagge (*op. cit.*) remarks, "It is a point of great importance that the development of sarcomatous growths

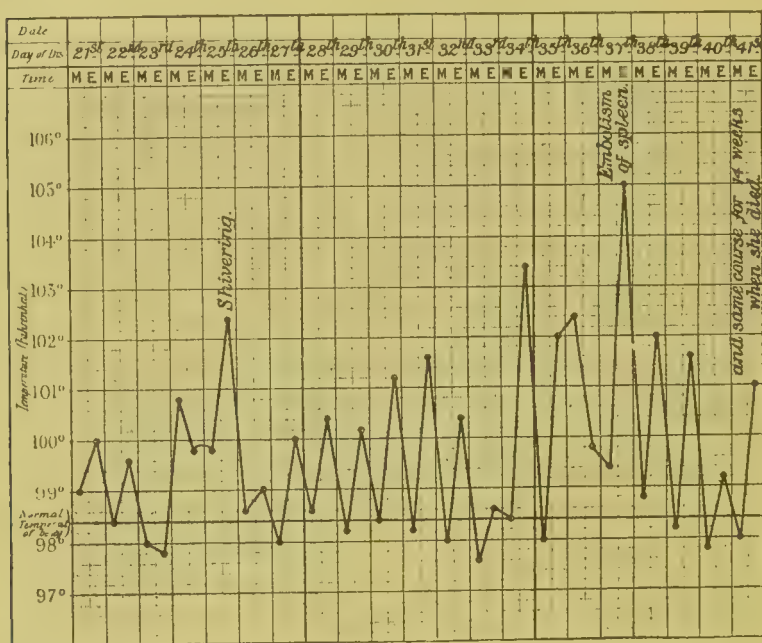


Fig. 107.—**MALIGNANT OR ULCERATIVE ENDOCARDITIS** in a female patient, at. 42, who was admitted to the Paddington Infirmary in the year 1890. The 3 weeks shown illustrate the course of the temperature over a period of 17 weeks, when she died. The chart of another case will be found on p. 75.

in the interior of the body is sometimes accompanied with symptoms such as by no means suggest the real nature of the cause." He mentions cases in illustration. *Symptoms.* The course of the fever varies somewhat, but it is usually of a diurnally intermittent character. This is accompanied by rapidly advancing emaciation and pallor, which, occurring without any other clinical features, give rise to considerable obscurity. "In some instances the chief thing of which the patient complains is pain, which may either be fixed, in certain parts, or widely diffused." Other symptoms which may be met with are purpura, spongy gums, epistaxis, hæmaturia, and bleeding from the mucous membranes.

*Diagnosis.* These cases appear to resemble Tuberculosis in the course of the temperature and their duration. The illness may extend over several months. The pains in the limbs and the perspiration give a resemblance to rheumatic fever. There seems to be a tendency to infect the bones, and a very thorough examination of these should always be made.

*Etiology.* The cases mentioned by Fagge were aged 16, 25, 28, 38, and 46, all being males. The prognosis is of course of the gravest kind, and all of Fagge's cases were fatal; some were not diagnosed during life. As regards treatment, Coley's fluid might be tried.



*THE GENERAL TREATMENT OF MICROBIC DISORDERS.*

Remedial treatment has for the most part been given under each disease, but there are some important matters relating to all fevers in common which must now be referred to, viz., **Immunisation, Serum Therapeutics, Notification and Isolation, Disinfection, Diet**, and the treatment of **Pyrexia** and **Hyperpyrexia**. In the first two of these we find ourselves on the threshold of discoveries which are revolutionising the methods of treatment and prevention of infective disorders.

§ 388. **Immunity.**<sup>1</sup> Before entering on the treatment of microbic disorders by inoculation (serum therapeutics) it will be desirable to discuss briefly the meaning of the terms Natural Immunity, Acquired Immunity, Artificial Immunity (Active and Passive), and Dual Immunity. A state of IMMUNITY is that in which an individual or an animal is more or less protected against contracting a certain disease. There is no such thing as absolute immunity; it is only a question of dose as compared with susceptibility. Given a large enough dose of the virus, and even a hen may develop tetanus. Some of the greatest achievements in preventive and remedial medicine have recently been reached in this domain. It was a triumph of this kind which Jenner achieved, though by purely empirical means, in the latter part of the last century (1796), in the prevention of small-pox by means of vaccination. After an interval of three-quarters of a century, this important department of medicine has been enriched by the researches of Pasteur in hydrophobia and anthrax, Koch in tubercle, Loeffler (1888), Fraenkel (1890), Behring, Roux and Kitasato in diphtheria, tetanus, and pneumonia, Haffkine in plague and cholera, Ross in malaria, Wright in enteric, and many others too numerous to mention. Some idea of the latent power for good in these researches may be grasped when it is remembered that the case-mortality of one disease alone (diphtheria) has been reduced from over 30 to under 15 per cent.

a. **NATURAL OR INHERENT IMMUNITY** is that form of immunity which a human being or the animal possesses at birth (or acquires during its growth), either by virtue of its species, race, or

<sup>1</sup> Dr. George Dean, of the Jenner Institute, has very kindly read the manuscript of §§ 388 and 389.

individual peculiarities. Thus, different animals are susceptible to various infective disorders in different degrees—hens are practically immune to tetanus, goats, sheep, and rats to tubercle. Again, certain races are immune to certain diseases—the negro is said to be immune to yellow fever.<sup>1</sup> Certain races appear to become after many generations relatively immune to some diseases, *e.g.*, measles among Europeans is now a very mild disease, but when it was accidentally introduced to the Fiji Islanders it became a devastating plague. Finally, certain individuals, and certain families, are more prone to contract infective disorders than others. I know of one family where 3 out of 6 members have had scarlatina twice, and some of the other infectious fevers more than once. Different families certainly vary in their susceptibility to infectious disorders.

b. ACQUIRED IMMUNITY is produced by contracting a disease in the usual way by infection. It has long been known that one attack of certain of the infectious disorders confers on the individual immunity from a second attack. The degree of immunity from second attacks differs considerably in the different diseases, and roughly one may make three groups, thus :—

a. One attack of the disease confers very strong protection against a second attack in Varicella, Scarlatina, Small-pox, Syphilis, Pertussis, Enteric, Dengue, Typhus, Yellow Fever, Mumps, and Whooping Cough. In the first five of these, and probably in the others also, the immunity lasts practically for a lifetime.

β. One attack confers only a moderate degree of immunity in measles, pneumonia, and diphtheria.

γ. Some confer immunity for only quite a short period, namely, erysipelas, cholera, dysentery and influenza. However, it seems probable that *all* infective disorders confer upon the individual a certain amount of immunity, for a short time at any rate.

From these facts the question naturally arises, cannot a mild attack be produced by inoculation of the infected material from a patient so as to secure immunity? And this question was successfully answered in the case of small-pox, which was so extensively

<sup>1</sup> Compare footnote to § 373.

inoculated in the early part of the 19th century, and which was finally forbidden by law after vaccination became compulsory.

c. ARTIFICIAL IMMUNITY. Now, what is it that creates this quality of immunity in an animal or an individual? and can this immunity be more scientifically produced? These are questions upon which pathologists have speculated for many years; but it was not until bacteriology had become a science that a solution of them seemed possible. It is now firmly established that all infective disorders, indeed the great majority of diseases attended by pyrexia, are due to the presence in the body of minute living organisms, namely, microbes or bacteria, most of which can be cultivated outside the body. Some of these measure no more than  $\frac{1}{10000}$ th of an inch. It is these minute organisms which constitute the infection or germs of the several disorders. Each disease has its own particular species of microbe, which has special qualities as to size, shape, growth, life-history, pabulum, virulence, and so forth; though the qualities of some (*e.g.*, the microbe of small-pox, scarlatina, and measles) have not yet been worked out. (2) Koch's four criteria, which identify a particular microbe as the specific cause of a disorder, are so important that they may be repeated here. (a) The constant presence of the microbe in all cases of the disease; (b) it can be cultivated outside the body, the cultures having constant properties; (c) these cultures when inoculated into animals can produce in them the same disease; and (d) the same microbe can again be recovered from the local lesion or from the body of the animal after death.

a. *Passive Artificial Immunity.* It is now known that the clinical manifestations and lethal effects of these organisms are chiefly, if not altogether, due, not so much to the microbes themselves as to the chemical products, the "TOXINS," which they evolve. The toxins and microbes can, moreover, be separated from one another by filtration.

The answer to the question "On what does immunity depend?" has been differently answered at different times. At one time it was believed to depend upon the presence in sufficient abundance of phagocytes to devour the microbes. Another theory was that an attack of the disease exhausted the store of pabulum

necessary for that particular organism which was to be found in the blood. Another theory was that the microbes left behind them something which rendered the soil unsuitable for the future growth of that particular microbe. None of these were found to be quite correct; but it has now been proved that the introduction of a particular kind of microbe or its toxin into the fluids or tissues of an animal sets up a chemical reaction in the body by means of which a chemical substance (which we call ANTITOXIN) appears in the blood, which more or less neutralises the toxin and prevents its lethal action. It is the presence of this antitoxin, or the faculty of again producing it on stimulation, after the bacteria have disappeared, which gives to the individual immunity from the infection of that particular disease. It follows therefore that if a particular microbe or its toxin can be injected into an animal in such doses as do not kill it, beginning with small doses and gradually increasing them, the blood serum will then be found to contain the antitoxin which antagonises the virus of the disease. In the case of diphtheria, for instance, this has been accomplished by the subcutaneous injection, at intervals of a few days into a horse (chosen chiefly because of the large quantity of serum available) of gradually increasing doses of the virus of that disease, *i.e.*, the diphtheria microbe grown in bouillon or some other suitable medium.<sup>1</sup> In this way it was found that the animal would gradually tolerate enormous doses of the virus—doses  $\frac{1}{1000}$ th part of which would have killed the animal before the immunisation was commenced. In short, the animal in this way had become highly immunised. Next, it was found that if a relatively small quantity of the serum of such an animal were injected into other experimental animals, it protected them against many times the lethal dose of the virus. Finally, it was found that if a small dose, say 10 or 20 c.c. of the immunised horse's serum were hypodermically injected into a human being, it rendered him similarly immune to the disease. Moreover, it was found that if the serum were injected even after the individual had contracted the disease (if given at a sufficiently early stage) it would cut the disease short, and prevent the lethal consequences. This kind

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<sup>1</sup> Some of the methods of introducing the virus are mentioned below under Active Immunity.



of artificial immunity—produced by the injection of serum from an immunised animal—is called *Passive Immunity*.

The explanation of all this is not yet by any means certain, but Ehrlich's theory is the one now generally believed, which is as follows. The microbic *toxins* produce their lethal effects by combining with some constituents of the cells of the body for which they have a special affinity. But the corresponding *antitoxin* has a stronger affinity for the toxin than the latter has for the body cells, and therefore the antitoxin acts by combining with the toxin and thus preventing the latter from doing harm.

*β. Active Artificial Immunity.* We have seen that Passive Immunity is that kind which is produced by the injection of the serum of an immunised animal; it is, as it were, a “borrowed” immunity. The term Active Immunity is applied to that kind of exemption which is acquired by the actual introduction of the microbe or its products into an animal or person whom it is desired to immunise. In the latter instance the individual manufactures his own antitoxin; in the former he receives the antitoxin manufactured in the body of another.

As long ago as 1880 Pasteur began his brilliant series of experiments showing that if animals were first inoculated with microbes weakened by age, heat, or exposure to the atmosphere, and were subsequently inoculated with the most virulent and actively growing cultures of the same organism, they had a very mild attack of the disease in question.

The following are some of the methods by which active immunity may be produced; the chief object being to attenuate the virus down to a suitable degree.

1. By taking some of a virulent living culture diluted with sterilised saline solution and injecting a small *non-lethal dose*. This is followed by constitutional symptoms; and then when these have subsided a second dose is injected, and then a third, and so on. The blood normally possesses a certain amount of bactericidal power. It is only when too large a dose, that is to say, too many bacteria, are introduced that they become lethal; and, therefore, it has been found possible to immunise an animal by a series of injections of non-lethal doses of the microbe in question.

2. By the injection of a living culture of microbes, the virulence

of which has been *attenuated in some way*, either by growing it in the presence of a weak antiseptic, or in the presence of oxygen, or in a current of air. The virulence of some microbes may be attenuated also by passing them through one species (a less susceptible species, for instance) of animal, which attenuates it for another species; or, again, by growing the culture at an abnormal temperature.

3. By the injection of *dead microbes* (killed by heat, for instance) in a series of gradually increasing doses.

4. By filtering a bacterial culture the bodies of the microbes are removed, and then the toxins alone, which remain in solution, may be injected into the animal.

5. By injecting a combination of 3 and 4.

In these various ways *active immunity* may be produced in an animal or human being. And the blood serum of such an animal, when injected into another animal, is capable of producing passive immunity in the latter.

d. DUAL IMMUNITY. (Antibacterial and antitoxic immunity.) Further researches resulted in a most important discovery, namely, that there are two kinds of protection, depending on the fact above mentioned, namely, that two things are necessary to produce the disease—(i.) the microbes which convey the disease, *i.e.*, the infection; and (ii.) the toxins evolved from them after entering the body of the animal, and which give rise to the lethal effects. Thus, for instance, Haffkine found in cholera that by injecting the bodies of the microbes, an anti-infective immunity was produced which protected the person against taking the disease. But if by chance the disease were contracted, the symptoms of the attack would be as bad and as fatal as ever. He then argued that if some of the toxin were also injected, along with the dead microbes, the severity of a chance attack would be considerably modified. It is hoped to accomplish this double protection in the case of plague, cholera, and typhoid.<sup>1</sup> At any rate it will be seen that

<sup>1</sup> Haffkine's methods for the immunisation of the plague are as follows: He grows the plague microbe in a liquid medium (? broth) in such a way and for such a long time (5 or 6 weeks) that, as results,

(i.) the plague toxin is produced in great quantity;

(ii.) the plague microbes undergo deterioration.

Then he heats the mixture to 65° or 70° C. and so kills the microbes. This preparation containing virulent toxin and dead microbes, injected into a healthy person, sets up moderate constitutional symptoms in two to five days. During that time the dead microbes

there may be two kinds of immunity or protection : (i.) an *anti-infective*, or antibacterial, immunity, produced by the inoculation of the microbe, dead or attenuated, which only prevents from infection ; and (ii.) an *antitoxic immunity*, produced by the inoculation of the toxin of the disease, which modifies the severity of an attack. It follows, however, that the latter, if it completely antagonises the toxins, affords adequate protection in both respects, because the microbes are deleterious only by their toxins. At any rate an antitoxic is relatively more valuable than an anti-infective protection.

The above refers to Active Immunity. In like manner the sera

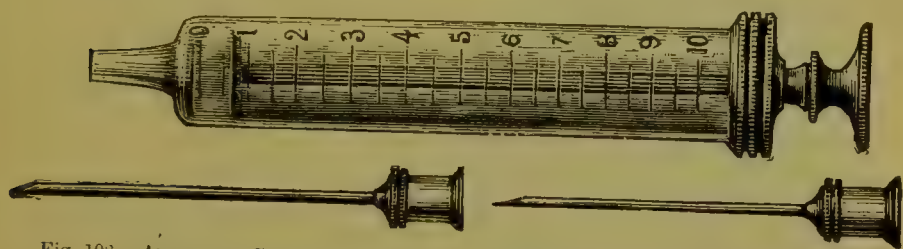


Fig. 108.—ANTITOXIN SYRINGE. The chief qualification of an *antitoxin* syringe is that it shall be capable of thorough asepsis and that the parts can be separated for that purpose ; the piston head generally being of asbestos. Another qualification is that it shall be capable of containing the whole of one dose. The syringe here figured contains 10 c.c. The dose of a *bacterial vaccine* is generally much smaller, and an ordinary hypodermic syringe, if capable of being thoroughly aseptified, will do. It is best, if possible, to boil the parts. Failing this, thorough soaking in strong carbolic solution and afterwards thoroughly washing with sterilised water, will serve the purpose.

used to produce Passive Immunity fall into two groups. Anti-streptococcic and anti-pneumococcic sera are illustrative of anti-infective sera ; while anti-diphtheritic and anti-tetanic sera are illustrative of antitoxic sera. Some processes of inoculation, like the anti-plague (Haffkine) and anti-typhoid (Wright), probably confer dual immunity.

§ 389. Remedial immunisation, or serum therapeutics.<sup>1</sup> It now remains to apply the principles enunciated above.

GENERAL PROCEDURE. 1. It is important to treat the case early, and to treat it boldly. Small doses may result in loss of valuable time. 2. The skin must be washed and every possible aseptic precaution adopted. 3. A special syringe is desirable, though any good thoroughly sterilised

have set up changes which result in the formation of an anti-infective serum, and the toxin has resulted in the formation of an antitoxin serum, and thus the patient is completely immunised in both directions.—*The Lancet*, June 24, 1899.

<sup>1</sup> This latter title, though in common use, is not altogether suitable, because immunity concerns the cells as well as the fluids of the body, and "serum" (the vehicle of passive immunity) is being largely replaced nowadays by prepared cultures and other means of conferring active immunity.

syringe may be used at a pinch. The best form is one in which all the parts can be taken to pieces and thoroughly boiled, because this is the best method of rendering it aseptic (Fig. 108). 4. If more than one dose has to be given it is advisable to make the injection into a different situation each time. The most usual sites are between the shoulders, the loin or buttock, the front of the abdomen, or the back of the arm. 5. Various skin eruptions of an urticarial or erythematous type may follow the injection of any serum.

The special methods for each disease are as follows, brought up to date, though it must be remembered that fresh discoveries are being made every day.

I. DIPHTHERIA.<sup>1</sup> An antitoxic serum has been in the market since 1895. When given early enough and in large enough doses, it has

been found to be of the greatest value as a remedial agent for patients suffering from the disease (see comparative mortality, § 366). It has also been used as a preventive, but it is for this purpose of only limited value; it confers immunity for a few weeks only. Some years ago *The Lancet* Commission tested several antitoxins in the market and found many inefficient. The antitoxin made at the Jenner Institute is sold through Allen and Hanburys.

The *contra-indications* for its use are given in § 366.

*Method.* The remedy should be used as early as possible in the disease. A dose of at least 4,000 units<sup>2</sup> should be given as early as possible, and repeated in half-doses

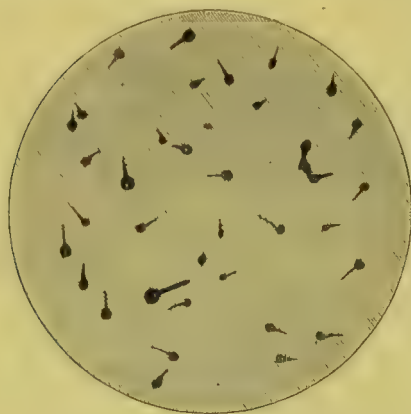


Fig. 109.—TETANUS BACILLUS.  $\times 1,000$ . Cover-glass preparation. Gentian violet. Photomicrograph by Mr. Frederick Clark.

every 24 hours until the exudation is obviously separating. Avoid injecting more than 20 c.c. at one place, if possible.

*Effects.* In the course of 24 hours there should be an improvement in the patient's symptoms, the membrane ceases to extend or perhaps begins to loosen, the swelling abates, and the rhinorrhœa is diminished. Occasional effects are urticarial or erythematous eruptions, additional rise of temperature or joint pains and swelling.

II. TETANUS (Fig. 109). Of late years the mortality from tetanus has been greatly reduced by the use of an antitoxic serum. The tetanus antitoxin was first prepared by Behring and Kitasato. There is no superiority in the French antitoxin over the German and English.<sup>3</sup>

*Contra-indications.* The longer the time that has elapsed between the infection through a wound and the injection of the serum the worse

<sup>1</sup> Dr. F. F. Caiger has kindly revised this subject to date, March, 1901.

<sup>2</sup> Behring's serum is at present the strongest in the market—3,000 units to 5 or 6 c.c. This same serum is now prepared at the Jenner Institute, and sold through Allen and Hanburys.

<sup>3</sup> It is prepared in large quantities at the Jenner Institute, and procurable from Allen and Hanburys, and elsewhere.



is the prognosis. It is usually too late to give the serum when tetanic spasms have appeared; yet cases are on record with recovery even after marked spasms had set in.

*Method.* In a decided case of tetanus 100 c.c. of the antitoxin should be injected within 24 hours, at different sites, in five doses. If there be no improvement next day, give it again, and continue with daily injections of about 20 c.c. Roux has found that the serum is considerably more potent by injecting directly into the cerebral tissue after trephining, and advanced cases have been saved by this means.<sup>1</sup>

*Effects.* The patient should sleep well on the night following the injection, and if fever is present the temperature should fall considerably the next day.

III. SEPTICÆMIA and pyæmia (including Erysipelas, Malignant Endocarditis, and Puerperal fever). The pyogenic (pus producing) organisms are capable of producing the septic intoxication we call septicæmia—streptococcus, staphylococcus, *b. coli communis* (in some circumstances), etc.—and the antitoxin of one will not act upon another. The antistreptococcic serum is an antibacterial serum which destroys the streptococcus present in the blood in the most typical form of septicæmia. It can be obtained from the Jenner Institute.

*Contra-indications.* This serum being useless, apparently, to cure a septicæmia due to the presence of any pyogenic germ other than the streptococcus, it is recommended first to examine the blood for micro-organisms. This is done by spreading some blood obtained from the patient (see § 351) on agar plates and examining the cultures.<sup>2</sup>

*Method.* As in all cases treatment should be commenced early, and since the case may be one of mixed infection, some do not consider it desirable to wait for a bacteriological report. Start with 20 c.c. and repeat once or twice daily as long as high fever or rigors continue.

*Effects.* In successful cases there should be an almost immediate fall of temperature and improvement in the condition of the patient. Failure is probably due to a mixed infection, with staphylococcus, etc.<sup>3</sup>

IV. ENTERIC FEVER (Fig. 110). In 1896 Wright, of Netley, introduced a method of *preventive* inoculation against Typhoid fever.<sup>4</sup> Agar cultures of typhoid bacilli are transferred to broth, and killed by heating at 60° C.



Fig. 110.—TYPHOID BACILLUS. Cover-glass preparation showing flagellæ.  $\times$  about 1,000. Loeffler's method. Photomicrograph by Mr. Frederick Clark.

<sup>1</sup> Method of intracerebral injection is given in detail by Dr. Semple, *B. M. J.*, Jan. 7, 1899.

<sup>2</sup> There are numerous pyogenic organisms the precise clinical and pathological effects of which have not yet been differentiated. The clinical effects of some of them appear to be interchangeable, as when the infection of erysipelas or scarlatina conveyed to a puerperal patient may give rise to all the symptoms of septicæmia.

<sup>3</sup> Mitchell Bruce, B. M. A. discussion, *Lancet*, 1898, ii., p. 515, and other articles in the journals of the past few years.

<sup>4</sup> Wright, *Lancet*, 1896, ii., p. 807, and *Brit. Med. Journ.*, 1897, i., p. 250.

This fluid is injected ; the strength of the doses being determined by experimental inoculation of guinea-pigs. Single doses are supplied in asepticised "vaccine" tubes, with full directions as to the method of use.

*Effects.* The immediate effects of a full dose are loss of appetite, faintness, slight fever and restlessness ; and in 24 to 48 hours the patient is well again. Locally there may be œdema : in some cases this is extensive, and Wright has found that by giving large doses of calcium chloride before the injection the tendency to local œdema is overcome. The immunity conferred probably lasts a few months—the blood serum may give the Widal reaction for this period.

An *antitoxin* for administration during an attack of enteric is on trial. It is given when the fever is remaining continuously high, and when the usual treatment appears to fail.<sup>1</sup>

*Method.* About 5 to 10 c.c. of the antityphoid serum are injected, and the dose is repeated daily if the fever rises after the fall consequent on the first injection. More recently Wright has employed liquid cultures.

V. TUBERCULOSIS. (α) Koch first introduced a "tuberculin," made by filtered bacillus cultures. This product is now known as the "old tuberculin," and is used solely for diagnostic purposes, for it only produces a reaction in an individual when he has tuberculosis in some part of his body. In this country, it is used chiefly for animals.

*Method.* Half a milligramme is injected, and the temperature is taken every 4 hours. If during 2 days there is no rise 2 milligrammes are given, and the temperature taken for 2 days again ; then 5 milligrammes, and the temperature again taken. If there is no elevation of temperature at any time the case is not one of tuberculosis.

(β) Koch has introduced a "*new tuberculin*" within the last few years, which promises much better remedial results than the old.

*Contra-indications.* The new tuberculin should not be used in cases where the temperature is over 105°. No dose should be given which causes a rise of temperature of over half a degree. The most suitable cases are (1) those in which the temperature has not gone above 99·5—100° F. ; (2) where the lesion is of small extent and only in one lung ; (3) cases where no mixed or superadded infection is present (§ 98).<sup>2</sup>

*Method.* Inject hypodermically  $\frac{1}{500}$  mgrm. diluted in normal saline solution, and increase the dose every second day until 20 mgrms. are tolerated. The immediate effects of a suitable dose are very slight. If the temperature rises over half a degree the dose has been too large. It is necessary at first for the patient to lie up, but in favourable cases, after a few doses, he can, if a hospital patient, go to hospital for injection each week, and return to work next day.

VI. HYDROPHOBIA. The Pasteur treatment of hydrophobia has obtained a world-wide reputation. Rabbits are inoculated with the virus of hydrophobia, and their spinal cords are taken out and dried. The longer these are allowed to dry, the more attenuated is the virus contained by them. Emulsions are made of the cords, and these are injected into the patient. Weak cords, which have been dried for 15 days, are first employed, and the virulence of the cord employed is gradually increased for ten days.

<sup>1</sup> Cases are narrated in *Brit. Med. Journ.*, 1897, i., pp. 259, 578.

<sup>2</sup> The most usual infection found with tuberculosis is a pyogenic one, due to septic absorption from the lungs or elsewhere. In such cases there is profuse expectoration of purulent sputum, the temperature is high and often of wide range. Heron, *Disc. at Med. Soc., Lancet*, 1899, ii., 1018.

*Contra-indications.* Treatment must be commenced as early as possible after the date of infection. The danger to be avoided in the treatment is a too rapid increase in the strength of the virus.

*Method.* The practical points for the practitioner are—(i.) to cauterise the wound at once; and (ii.) to send the patient to the Pasteur Institution, Paris, taking with him, packed in ice, the head of the animal which bit him. Thus it can be ascertained whether the bite was dangerous or not.

The *results* of this method of treatment are very encouraging. The ordinary mortality of bitten patients, before the institution of this treatment, was about 16 per cent.; but from 1886 to 1895 (17,337 cases) the mortality was 0·48 per cent.<sup>1</sup> A serum treatment of hydrophobia is at present on trial.

VII. PLAGUE. The serum treatment of plague is still on trial in India. Yersin's<sup>2</sup> or Lustig's<sup>3</sup> serum is employed. It appears to be an antibacterial serum, and is attended with considerable success. As so few cases can be obtained in time for this treatment, Haffkine<sup>4</sup> has introduced a method of preventive inoculation of attenuated cultures which it is believed confer immunity for about one year. There is a prospect of this being both *anti-infective* and *antitoxic*.

*Contra-indications.* The treatment must be commenced on the first day of the illness, because the course of the disease is so rapid and severe that later administration cannot check its progress.

*Method.* From 20 to 40 c.c. are injected daily for one to ten days, according to the nature of the case.

*Effects.* Cases have recovered in two days when the treatment was commenced on the first day of illness. In such cases the mortality has been greatly reduced.

VIII. CHOLERA. A cholera antitoxin has been introduced, but is still on trial. Haffkine has been able to confer immunity for a year by inoculating two or three times with attenuated cholera cultures; then with more exalted virus. The results were encouraging; fewer people were attacked; but those who were attacked did not have a modified or milder form of the disease. Hence he has more recently attempted to produce an inoculating material which shall contain antitoxic properties as well as antibacterial.

IX. SNAKE POISON. Calmette introduced an antitoxic serum for the poison of snake bite, which is known as antivenene. The serum can be kept for a long period in a tropical climate without losing its properties.

*Method.* Inject as soon as possible after the bite at least 10 c.c. of Calmette's antivenene, and repeat the dose some hours later.<sup>5</sup>

*Effects.* The patient recovers very soon if the injection is given before unconsciousness or paralysis set in. Even if given when respiratory paralysis threatens this dangerous symptom may not ensue, and the paralysis of the limbs usually disappears in less than two days.

X. PNEUMONIA. The serum treatment of pneumonia has as yet been tried chiefly on animals. In animals remarkable results have been

<sup>1</sup> "Bacteriology," Muir and Ritchie, p. 472, 1st edition.

<sup>2</sup> *Lancet*, 1897, i., p. 604; and ii., p. 1546.

<sup>3</sup> *Lancet*, 1897, ii., p. 1361.

<sup>4</sup> *Lancet*, 1899, June 24.

<sup>5</sup> Cases narrated *Brit. Med. Journ.*, 1899, ii., pp. 1432, 1732, and elsewhere.

obtained. Thus 1 c.c. of Pane's serum protected a rabbit against 3,000 living doses of a living culture of the pneumococcus.<sup>1</sup> Good results are expected from this method of treatment in the human being.<sup>2</sup> The serum of a patient who has recovered from pneumonia protects, to some extent, rabbits from the pneumococcus. This serum is probably anti-bacterial. It appears that there are several varieties of pneumococci and that one kind does not protect against another.

XI. ANTHRAX. Animals have been inoculated with increasingly virulent doses of anthrax cultures, and the results are encouraging as a preventive. The serum of the inoculated animals is probably anti-bacterial.

XII. YELLOW FEVER. Sanarelli, who isolated the microbe of Yellow Fever in 1897, has apparently succeeded in manufacturing a serum which is effectual against the microbes, not against the toxins, of the disease.<sup>3</sup>

*Contra-indications.* It is useless to give the serum when the renal or nervous systems are affected, as evidenced by anuria and delirium. The serum must be given early, about the 1st or 2nd day, before toxins in any quantity have had time to be elaborated.

*Method.* Large doses are injected, and repeated until signs of recovery are present. One of Sanarelli's cases recovered after 80 c.cm. had been injected. After each dose the temperature fell, and if albuminuria was present, it disappeared.

§ 390. **Notification and Isolation.** Two duties are laid upon the medical practitioner in cases of the commoner infectious maladies. 1. NOTIFICATION of the case to the medical officer of health of the district in which the case arises. The notifiable complaints in most districts are scarlatina, diphtheria, "membranous croup," enteric fever, and "continued" fever, small-pox, cholera, erysipelas, typhus, relapsing fever, puerperal fever, and plague (measles, varicella, and phthisis are *voluntarily* notifiable, and the public ambulances may be used for the first two). A medical man is bound under a penalty of 40 shillings to notify any of the maladies named "immediately on becoming aware" of its existence. 2. REMOVAL of the patient to a fever hospital is compulsory, unless the parents or guardians can make *proper* and *adequate* arrangements for the isolation of the case at home. In some places the removal is superintended by the M.O.H. In the metropolitan area the medical practitioner should at once communicate with the central office of the Metropolitan Asylums' Board, Victoria Embankment, E.C., when an ambulance will promptly be sent for the case. Their telegraphic address is "Asylums' Board, London," and the particulars required to be sent are Name, Address, Disease, Age and Sex of patient, and Severity of case.

It is far better for the patient and for his relations that he should be removed to a properly organised Fever Hospital; but to isolate a patient at home hang a sheet, constantly wet with carbolic solution (1 in 20) across the door or passage. Carpets, curtains, and superfluous furniture should have been previously removed. Books and articles in use must be such as can be afterwards burned. Ventilation must be carried out as described below. The nurse in charge of an infectious case should wear a washable dress when on duty, and should hold no communication with others, nor should she go out of doors without having first changed her wearing apparel, and, if possible, taken a bath. An airy, quiet room *at the top of the house*, having cubic space of about 12 × 12 × 10 feet, is desirable. The air in this space requires to be changed 3 or 4 times in every hour. Only the furniture in immediate use should be allowed to remain. The carpet should be taken up, and all

<sup>1</sup> Washbourne, Med. Soc. Report, *Lancet*, 13th Oct., 1899.

<sup>2</sup> A successful case is narrated by Dr. Harnett in the *Brit. Med. Journ.*, 1897, i., p. 1279.

<sup>3</sup> *Brit. Med. Journ.*, 1898, i., p. 1028.



stuffed furniture removed. The bedstead should be so placed as to be accessible on both sides. The temperature, read on a thermometer suspended near the bed, and away from draughts, should be 60° F.

VENTILATION must be ample in fever cases because of the danger of mixed infections. There are reasons for believing that the tonsils are sometimes the portal for infection, and that, perhaps, is the reason why mixed infections are more apt to arise in cases of scarlatina when there is not free ventilation and sufficient cubic space. This partly explains the higher death-rate from infectious diseases when overcrowding occurred in former days. The direction of the wind should be constantly noted, and to avoid draught, the windows or ventilators opened on the side of the room away from the wind. A "sash board" is an excellent contrivance for avoiding draught. It should be about 6 to 8 inches broad, and fit across the bottom of the window so that the lower sash can be raised without a visible opening, and then ventilation takes place behind the sash board, and also in the middle of the window, the air in both cases being directed upwards. The chief principle involved in all ventilation is that the current of air always takes place from a colder to a hotter medium—usually, therefore from outside to the inside of a room. The chimney, when the fire is alight, is the only reliable *exit*. Make the window your *inlet* in preference to the door.

§ 391. **Disinfection and Prevention.** Before describing the means employed for disinfection, it is necessary briefly to describe the way in which microbic disorders are propagated. Since bacteriology has become a science, great advance has been made in this direction. There are 3 principal ways by which infection is conveyed—by the *air*, by *water*, or other ingesta, and by direct *contact* or inoculation—and microbic diseases may be thus classified.

a. As regards the *air-borne* group, there is considerable variation in their infectivity, also the distance to which the contagion in an active state may be carried through the air; for instance, erysipelas and typhus probably do not spread beyond a few feet, but small-pox and scarlatina may spread for many yards, some say the former spreads to a distance of a mile or more.<sup>1</sup> Air-borne diseases can also be conveyed by furniture and other articles in common use. The portal by which most of these diseases enter the system is generally believed to be the lungs; but certain facts lately observed point to the tonsils, throat, and nose as possible channels for their introduction. Some of this group may be conveyed by milk, and it is possible that other ingesta may become contaminated by the contagia of these diseases. The air-borne diseases are as follows: Varicella, Scarlet Fever, Small-pox, Measles, Rubeola, Typhus, Dengue, (air-borne or miasmatic), Diphtheria, Erysipelas, Plague, sometimes Influenza, Mumps, Rheumatic Fever, Whooping Cough, Yellow Fever. Pulmonary tuberculosis arises from the inhalation of contaminated particles.

b. The *water-borne* group only comprises three diseases, viz., Enteric Fever, Cholera, Dysentery. Two facts form the basis of the propagation and prevention of these diseases—(1) All matters coming from the patient's bowels and stomach are infective, in enteric the urine also; and (2) to produce the disease the virus must be introduced by the mouth into the alimentary canal.

c. The *third group* comprises disorders the infection of which must be introduced into the blood or tissues of the body in order to produce the disease either by means of a wound or a scratch which may perhaps have escaped notice. Our profession pays a penalty every year to this group of disorders when, perhaps, some overworked practitioner is called to the bedside of a *syphilitic* lying-in woman, and forgets to examine the margins of his finger-nails, where some crack or unsuspected scratch will be the means of the introduction of the syphilitic poison. Some of these disorders were formerly described as *miasmatic*, i.e., dependent upon some

<sup>1</sup> Some valuable data on this question were collected by the author from the Warrington small-pox epidemic, 1901–1902. Appendix to the Report of the Roy. Comm. on Vaccination

meteorological, telluric or climatic influence, which we did not understand. *Ague* is an example of these diseases, but it is now known to be directly introduced into the blood of the patient by the bite of a mosquito. *Tetanus* and *plague* are other examples; tetanus is introduced through a wound or scratch which has become contaminated with the soil, plague is conveyed by rats. *Septicæmia* is due to the internal or external contamination of the blood current, and all kinds of dust probably contain pyogenic, i.e., septicæmic microbes. *Glanders* is contracted from horses by the contamination of a wound or scratch; and *Anthrax* is contracted by woolsorters and others who come in contact with the hides of animals containing the contagion, and thus inoculate a scratch or inhale the dust. *Tuberculosis* is placed under this group because it is sometimes undoubtedly inoculated into a wound giving rise to lupus vulgaris or verruca necrogenica on the hand. *Hydrophobia* must be inoculated, generally by the bite of an animal suffering from rabies. *Gonorrhœa* is conveyed either to the urethra or conjunctiva, but whether a breach of surface is necessary or not is not known.

It follows, therefore, that the procedure for disinfection differs somewhat in the case of air-borne diseases, water-borne diseases, and those introduced by the contamination of a wound or scratch.

(1) FOR AIR-BORNE DISEASES—

(i.) The *linen*, before washing, should be left to soak in carbolic solution (1 in 80). In any case, rather than leave clothes and linen exposed to the air, keep under water until they can be removed (a wineglass of carbolic acid to a gallon of water is roughly 1 in 80).

(ii.) *Clothes and bedding*. If a disinfecting oven (at a temperature of not less than 210° F. or more than 320° F.) or a steam-heated chamber at 212° F. is not available, they may be spread out in the room, and treated by sulphur (see below) or formalin spray. It is very doubtful if the fumigation of clothes by sulphur is of much use. *Washable articles should be plunged into a tub containing carbolic solution (1 in 80, vide supra), and then sent to the wash, when they should be boiled.*

(iii.) The *patient*, before returning to his friends, must have several warm baths and be well washed with carbolic soap. This is especially necessary in diseases where desquamation occurs.

(iv.) To disinfect the *room*. Close the windows and doors, and stop up all crevices. Melt some sulphur over a fire in a saucepan or small iron bucket, set it alight, and place it on an old tray in the middle of a room; then shut up the room for twenty-four hours. Use 1½ lb. sulphur for every 1,000 cubic feet; 3 lb. for an ordinary sized room. The fumes are very suffocating but they will not hurt anything if the air be dry, excepting brass, and this may be protected by smearing it over with vaseline. A whole house may be fumigated in this way from the basement, by closing the windows, stopping up the chimneys with newspaper, and opening the doors of communication. Nowadays it is recognised that a gaseous is much less thorough than a fluid disinfectant, and the walls should be washed with perchloride of mercury or saturated with formalin sprays.—*Lancet*, Aug. 13, 1898; B. M. A. Discussion, State Medicine.

(2) FOR WATER-BORNE DISEASES—

(i.) The *excreta*, if practicable, should be burned; if not, before being removed, they should be covered with chlorinated lime or carbolic solution (1 in 40, *vide supra*).

(ii.) The *under-linen*, towels, bedding, etc., must be boiled or treated very carefully as in air-borne diseases.

(iii.) All *drinking-water* should be boiled if there is the slightest suspicion of its being contaminated by leakage, soakage (however small) from cesspools, drains, or the reckless casting of slops, etc. Pasteur filtration is said to be even more efficacious than boiling the water.<sup>1</sup>

List of common disinfectants:—Extreme heat (200° F. or more and preferably moist); fumes of burning sulphur (SO<sub>2</sub>); chlorinated lime; chlorine, evolved from chlorinated lime by hydrochloric acid (spirits of salts); carbolic acid (a wineglass of carbolic acid to each gallon of boiling water is roughly 1 in 80 solution); formic aldehyde; permanganate of potash (Condy's fluid); chinolol; lysol; sulphate of iron; sulphate of copper; creolin; corrosive sublimate; terebene; thymol; eucalyptol; sanitas.

(2) Disinfection and the PREVENTION OF DISEASES INCLUDED IN OUR THIRD GROUP, differs in each individual case. Thus Septicæmia and tetanus almost ceased in surgical cases with the introduction of cleanliness and asepsis. Ague depends mainly upon mosquitoes, and the extermination of these is now engaging much attention.

<sup>1</sup> *The Lancet*, Sept. 16, 1899, p. 803.

§ 392. Diet in fevers is a question of great importance because all the secretions and excretions are so much diminished or modified in fever cases. It should consist mainly of milk and meat juices. At least  $3\frac{1}{2}$  pints of milk per diem may be given, fresh, sterilised if possible, or scalded (not boiled), in small quantities at a time; and it may often with advantage be diluted with half or a third of water, soda water, or barley water. Lime water may be used instead, if diarrhœa be present, or if there be constipation, a drachm of soda bicarb. to the pint. If milk is not well tolerated whey or cream may be given, or the yolks of eggs or egg flip. Beef-tea, chicken or mutton broth, about a pint in the 24 hours, should also be given, but as these do not contain all the constituents of the meat they may with advantage be replaced or supplemented by some of the many excellent modern substitutes (*e.g.*, Liebig's or Valentine's extract, Bovril, &c.). Some methods of preparing invalid foods are given § 214. Iced water is very agreeable, but it generally increases the thirst. Fresh lemonade may be advantageously substituted by mixing a drachm or two of bi-tartrate of potash, with a little sugar, to the pint of water.

§ 393. The treatment of pyrexia and hyperpyrexia comprises 6 indications—

1. *Heat production* can be diminished to some extent by means of drugs, known as antipyretics, such as antifebrine, antipyrine, and phenacetin. The first of these is, on the whole, most efficacious for reducing temperature, but it requires care on account of its depressing effect on the heart, and the reaction which follows some hours later. Quinine in full doses (say 5 grs. every 3 or 4 hours) may be given until the temperature comes down, or physiological symptoms are produced (singing in the ears, deafness, headache, &c.). Salicylates, especially in rheumatic affections, and aconite are also useful. Among the more familiar but less efficacious febrifuges and diaphoretics are liq. ammon. acet., pot. nit., sp. eth. nit., and camphor, also lemon drinks, dilute acids, and salines. Kairin is said to reduce febrile temperature very rapidly, but is apt to produce profuse sweating (which may however be combated by atropine), or shivering (which may be combated by quinine), or collapse, if the dose be too large. Parthenine (an alkaloid derived from *Parthenum hysterophus* (Linneus), has been known as a febrifuge for a long while to the country people in Havana, where its common name is Escoba Amarga. Febrifuge doses of 2 grammes may be given. It was tried with success in 80 patients by Dr. Ramirez Tovar.

2. *To aid the loss of heat* is a method of treatment called for in cases of hyperpyrexia (*i.e.*, when the temperature reaches above  $104.5^{\circ}$  F.), and especially if the drugs previously mentioned have failed, by means of the graduated bath, the wet pack, sponging, the application of ice-bags, or Leiter's Coil.

*The graduated bath.* Place the patient in a bath one-third full of water at  $90^{\circ}$  or  $95^{\circ}$ . Every five minutes reduce the temperature  $5^{\circ}$  until  $60^{\circ}$  is reached. If the patient's fever be not then reduced to  $100^{\circ}$  or lower, he may be left in a further quarter of an hour if his pulse be a fair strength. The pulse must be closely watched, and alcohol given if necessary.

*The Wet Pack.* Take off the night-shirt and superfluous bed-clothes, and place the patient on a blanket. Moderately wring a sheet out of ice-cold water and lay it along his side. Gently roll him over on to it and completely envelop him in it, head, and all, except the face; so that it is next his skin, without creases or air, between the legs and beneath the arms.

Cover these latter with wet towels. Then put two cradles over the patient, and blankets over all. Leave him thus packed for twenty to forty minutes, until his temperature, taken in the mouth, is reduced to the required extent.

*Tepid Sponging.* Lay the patient in a blanket and sponge him gradually all over with tepid water (about 75°). Do half the body at a time, the other half being covered up. Continue the process for twenty to forty minutes, until the fever is reduced.

*The application of ice* in large ice-bags for the head, chest, and abdomen has been used when other means are not available, but the weight of the bags and their localised application are objections to their use. *Leiter's Coil* consists of a specially made coil of metal or rubber tubing through which cold water is continually running. This coil may be applied to the head, abdomen, or chest. Neither of these two last methods are recommended for fever cases.

3. To *diminish the work done by the internal organs* is another means of combating pyrexia. This may be done by diet (*vide supra*), and by promoting the action of the skin and bowels, in order to relieve the kidneys. Saline purges fulfil the latter indication (F. 46, 51, 55, and 63 are useful).

4. In all fevers it is necessary to *watch the heart* very carefully, and, if necessary, to steady it by means of strychnine and digitalis, or to aid its flagging power by means of stimulants. The pulse should be examined several times a day in all fever cases, if only for this purpose.

5. *Symptomatic treatment* may also be necessary, but this has been dealt with in the preceding pages. The constipation must be relieved by calomel or saline purges; the thirst by lemon water in sips (not ice); and the headache by phenacetin, &c.

6. The last indication is to *watch for and treat complications* as they arise. The chief of these are (i.) cardiac (*vide supra*); and (ii.) delirium and insomnia. If the delirium be of the *raving* kind chloral and bromides should be given in full doses; if, on the other hand, it be of the *muttering* or typhoid variety, stimulants and ammonia are indicated. Insomnia may be relieved by the same treatment, and alcohol may be useful in this respect. (iii.) Pulmonary complications; (iv.) suppression or retention of urine; and (v.) collapse, are all dealt with elsewhere.



# FORMULÆ

(referred to as F. in the text)

## OF USEFUL PRESCRIPTIONS.

*The proportions given are those for one adult dose unless otherwise stated.*

### (1) BALNEUM ALKALINUM.

Add two large handfuls (8 ozs.) of common washing soda to 30 gallons of water at 95° F. The patient remains 20 minutes in first bath, and the time is gradually increased up to 45 minutes. Put to bed in blankets.

Valuable for chronic rheumatism—daily for 6 weeks. At first the pains are increased. Also useful for chronic eczema.

### (2) BALNEUM CREOL. VEL PICIS.

R Creolin ʒss to ʒij or Liquor Carbonis Deterg. . . . . ʒj to ʒiv,  
in 20 to 30 gallons of water, well stirred.

Useful for pruritus, prurigo, chronic eczema, and all itching affections.

### (3) BALNEUM SULPHURIS.

R Potass. Sulphurat. ʒij,  
Acid. Acet. dil. (or  
vinegar) . . . . . Oss,  
Warm water. . . . . 20 to 30 gals.

### (4) BALNEUM VAPORIS HYDRARG.

R Hydrargyri Subchloridi . . . ʒss.

Volatilise beside  $\frac{1}{2}$  pint of water for 20 minutes in a Lee's lamp. Seat the patient in a cane-bottomed chair. Tie the curtain round the neck, and put a blanket over the shoulders. Light the lamp, and place it beneath the chair. Leave the patient in from 20 to 40 minutes.

### (5) SHOWER BATH.

See that the shower is the required temperature<sup>1</sup> before placing the patient in the curtain, then turn on *suddenly*, because the chief therapeutic value of the shower is by its shock. It should not be given where marked heart disease exists. Valuable in hysteria and neurasthenia.

### (6) HOT-AIR BATH.

Remove the clothing and lay the patient in a blanket, adjust the wicker framework and cover it with three or four blankets, which should come up well under the chin. Light the torch under the chimney, and let it continue to burn until the patient perspires very freely. He may remain for fifteen or twenty minutes longer. The temperature inside the wicker work should be between 170° and 200° F. Valuable in renal disease.

Collunaria—Nasal douches. Should be used at a temperature of about 100° F. Half a tumblerful to be injected along the floor of each nostril night and morning. The patient should be directed *not* to blow the nose forcibly immediately afterwards.

### (10) COL. POT. CHLOR. COMP. (C. L. Throat Hosp.).

R Pulv. Pot. Chlor. . . . .  
Pulv. Boracis . . . . .  
Sodæ Bicarb. . . . . āā gr. xij,  
Pulv. Sacch. Alb. . . . . ad ʒj.

To be added to  $\frac{1}{2}$  tumblerful (ʒv) of water. Useful in all forms of chronic rhinitis.

<sup>1</sup> Cold 50° F., tepid 75° F.

(11) COLLUNARIUM AC. CARBOL.  
(vel BORACIS).

ʒj of the Glycerinum Carbol. or Boracis  
(B.P.) to 10 fluid oz. of water.

## (13) ENEMA HAZELINÆ.

Hazeline and water . Of each 1 oz.

Administered slowly by means of a  
glycerine syringe for internal piles.

## (14) ENEMA NUTRIENS.

- (a) Peptonised milk, 2 to 4 oz.  
(b) Strong beef tea, 2 oz., and meat  
juice, 1 oz.  
(c) One egg, 1 oz. brandy, 2 oz. strong  
beef-tea or 2 teaspoonfuls of  
meat extract; and 1 teaspoonful  
of Bullock's acid glycerin of  
pepsin; mix and divide into  
two enemata.

These may be administered alternately.

(15) GARG. ACIDI CARBOL. c Co-  
CAINA.

℞ Acidi Carbolici . . . ʒj,  
Cocainæ Hydrochlor. . . gr. viij,  
Glycerini Boracis . . . ʒss,  
Aquam Rosæ . . . ad ʒxiij.

For acute pharyngitis and laryngitis.

## (16) GARGARISMA ACIDI TANNICI.

℞ Glycerini Acidi Tannici . ʒj,  
Aquam . . . ad ʒj.

For relaxed throat and to check bleed-  
ing after tonsilotomy.

(17) GARGARISMA BORACIS COM-  
POSITUM.

℞ Pulveris Aluminis.  
Pulveris Boracis . . . āā gr. vijss,  
Tincturæ Myrrhæ . . . ℥ v,  
Mellis . . . gr. x,  
Aquam . . . ad ʒj.

## (18) GARGARISMA CHLORINI.

℞ Potassii Chloratis . . . ʒij,  
Acidi Hydrochlorici For-  
tioris . . . ʒj.

Cork and set aside for 5 or 10 minutes,  
then add—

Glycerini . . . ʒiv,  
Aquam . . . ad ʒxiij.

To be freshly prepared. A very  
prompt and efficacious remedy for scar-

latinal and diphtheritic sore throat, hos-  
pital sore throat, and follicular tonsillitis.  
For children it should be applied with a  
brush every 2 hours.

(19) GARGARISMA POTASSII CHLO-  
RATIS.

℞ Potassii Chloratis . . . ʒiss,  
Aluminis . . . ʒiss,  
Aquam . . . ad ʒx.

## (20) GLYCERINUM CARBOLICI FORTE.

℞ Acidi Carbolici . . . ʒiij,  
Glycerini . . . ʒj.

For applications in endocervicitis.

## (21) GLYCERINUM FERRI.

℞ Liquoris Ferri Perchloridi . ʒiv,  
Glycerini . . . ʒiss,  
Aquam . . . ad ʒj.

To paint the tonsils in recurrent tonsil-  
litis.

## (22) GUTTÆ AURIBUS.

℞ Plumbi Acetatis . . . gr. j,  
Tincturæ Opii . . . ʒj,  
Glycerini . . . ʒj,  
Aquam Rosæ . . . ad ʒj.

Used warm for inflammation or pain of  
the external auditory meatus.

Glyc. Ac. Carbol. (B.P.) answers the  
same purpose.

## (23) GUTTÆ DENTIBUS (Dr. Gaye).

℞ Olei Caryophylli . . . ʒss,  
Etheris . . . ʒiss,  
Tincturæ Opii . . . ʒij,  
Glycerini . . . ʒij.

To drop in a painful hollow tooth, or to  
apply on cotton wool. Washing out the  
mouth with warm carbolic lotion (1-100)  
often relieves toothache.

(24) INJECTIO HYDRARG. HYPODERM  
(Durham).

℞ Ammonii Chloridi . . . gr. viij,  
Hydrargyri Perchloridi . gr. xvj,  
Aquam Destillatam . . . ad ʒj.

Dissolve—℥ x contains  $\frac{1}{8}$  gr. Hydrar-  
gyri Perchloridi. Inject ℥ ij to ℥ v into  
the muscle of the buttock or shoulder  
every 2nd, 3rd, or 4th day.

Another formula consists of Hydrarg.  
Succinimide, 2 %, with Cocain. Hydroch.  
 $\frac{1}{2}$  %—dose, 20—30 ℥. It is less painful  
and equally efficacious.

(25) INJECTIO MORPH. CUM ATROPINÂ.

R Atropinæ Sulphatis . . gr.  $\frac{1}{2}$ ,  
Morphinæ Acetatis . . gr. viij,  
Sol. Ac. Carbol. (1 %) . . ad 3iv.

(M v contain  $\frac{1}{100}$  gr. and  $\frac{1}{8}$  gr. respectively.)

(27) INJECTIO PILOCARPINÆ.

R Pilocarpinæ Nitratis . . gr. v,  
Sol. Ac. Carbol. (1 %) . . M 100.

Dose 2—5 M. Rapid diaphoretic. M j useful to reduce arterial tension. Also used in nerve deafness, gr.  $\frac{1}{10}$  gradually worked up to gr.  $\frac{1}{4}$  daily, resting for two hours afterwards (Field, *B. M. J.*, 1889, vol. i., p. 471, and 1890, vol. i., p. 1125).

(28) INJECTIO STRYCHNINÆ Co.

R Strychninæ Sulph. . .  $\frac{1}{2}$ ,  
Atrop. Sulph. . .  $\frac{1}{8}$ ,  
Acidi Borici . . . 1,  
Acidi Carbol. (pur.) . . 1,  
Aquam Destillatam . . 100.

M v as often as required, to relieve the prostration and bodily discomfort in vascular dilatation, such as occurs in neurasthenia and morphia craving.

(30) LINCTUS COMMUNIS.

R Oxymellis Scillæ . . . 3ij,  
Syrupi Tolutani . . . 3ij,  
Tincturæ Camphoræ  
Compositæ . . . 3j,  
Aquam Destillatam . . ad 3j.

Dose.— $\frac{1}{2}$  a teaspoonful for bronchitic cough.

(31) LINCTUS SEDATIVUS.

R Liquoris Morphinæ Hydrochloratis . . M xl,  
Acidi Hydrocyanici Diluti M viij,  
Acidi Hydrochlorici Diluti M xvj,  
Glycerini . . . 3iv,  
Aquam Destillatam . . ad 3j.

Dose.—3j. Relieves the cough of phthisis. A teaspoonful in a wineglass of water may be sipped every five minutes until the cough is relieved.

LINIMENTA.

(32) LINIMENTUM AMMONIÆ Co.

R Olei Terebinthinæ . . . 5x,  
Liquoris Ammoniæ Fortioris . . . 3iv,  
Saponis Mollis . . . 3v,  
Camphoræ . . . gr. 80,  
Spiritus Vini Methylati 5ij,  
Aquam . . . ad 3x.

A stimulating embrocation, resembling Elliman's.

(33) LIN. BELLAD. CUM CHLOROF.

R Lin. Belladonnæ, Lin.  
Chloroformi . . . aa 3j.

Very valuable for lumbago and other rheumatic affections; sprinkled on lint. Do not cover with oil-silk, or it will blister. A very expensive liniment, which for hospital purposes can be made equally well with Lin. Terebinth instead of Lin. Chlorof.

(35) LOTIO ALKALINA OPIATA  
(Fuller).

R Potassii Carbonatis . . gr. 200,  
Liquoris Opii Sedativi . . M 400,  
Glycerini . . . 5x,  
Aquam . . . ad 5x.

Apply warm to painful rheumatic joints.

(36) LOTIO CALAMINÆ.

R Calaminæ . . . gr. xx,  
Zinci Oxidi . . . gr. xx,  
Glycerini . . . 5ss,  
Liq. Calcis . . . 3ijj,  
Aquam Destillatam . . ad 3j.

(37) LOTIO CALCIS CUM OLEO  
(Carron oil).

R Acidi Carbolicæ . . . Mx,  
Liquoris Calcis . . .  
Olei Lini . . . aa 3ij.

Mix well. Invaluable for burns.

(38) LOTIO CAPILLARIS.

R Tincturæ Cantharidis . . 5ss,  
Liq. Ammon. Fort. . . 5ss,  
Glycerini . . . 5ss,  
Aquam . . . ad 3j.

Rub into the head night and morning for baldness.

## (39) LOTIO CREOLIN.

Creolin ʒj—Oj of water, to wash skin diseases, before applying ointments.

If the skin is dry and harsh add Glycerin ʒj—Oj.

## (40) LOTIO EVAPORANS (vel FRIGIDA).

℞ Liquoris Ammonii Acetatis  
Spiritus Vini Methyлатi. . . . . āā ʒv,  
Aquam . . . . . ad ʒx.

Local application for acute gout, sprained joints, or headache. Invaluable for insect bites and stings. N.B.—Must not be covered by oil-silk.

## (41) LOTIO KEROSINÆ.

℞ Kerosene . . . . . ʒij,  
Olive Oil . . . . . ʒj.

For pediculi capitis. Destroys both insects and nits.

## (42) LOTIO PLUMBI CUM ZINCO.

℞ Lot. Plumbi Diac. Dil. . . . . ʒj,  
Zinci Oxidi . . . . . gr. xx,  
Glycerini . . . . . ʒss,  
Aquæ . . . . . ad ʒj.

Invaluable for acute eczema.

## (43) LOTIO HYD. CUM ACID. CARBOL.

℞ Hyd. Perchlor. . . . . gr. iss,  
Acid. Carbol. . . . . ℥ xx,  
Glycerini . . . . . ʒss,  
Sp. Vini Rect. (or Meth.) ʒj,  
Aquam . . . . . ad ʒj.

## (44) LOTIO SAPONATUS KALINUS (Hebra).

℞ Saponis Viridis . . . . . ʒj,  
Spir. Vini . . . . . ʒij.

Dissolve by gently heating, filter and add ol. berg. and ol. lav. aa ℥ xv. For cleansing the head from Schorrhœa.

## (45) MISTURA AMMON. CHLOR. CO.

℞ Ammon. Chlor. . . . . gr. xv,  
Liquoris Morph. Hydrochlor. . . . . ℥ v,  
Aquam Chlorof. . . . . ad ʒj.

4 tis horis in facial neuralgia.

## (46) MIST. APERIENS EFFERVESCENS.

No. 1.

℞ Sodii Bicarbonatis . . . . . gr. xxx,  
Sodæ Tartarata . . . . . gr. xl,  
Potassii Tartratis . . . . . gr. xl,  
Aquam . . . . . ad ʒj.

No. 2.

℞ Acidi Tartaric . . . . . gr. xxx,  
Aquam . . . . . ad ʒj.

## (47) MISTURA BROMIDI ET IODIDI.

℞ Potassii Iodidi . . . . . gr. v,  
Ammonii Bromidi . . . . . gr. xv,  
Spir. Ammon. Aromat . . . . . ℥ xv,  
Syr. Aurantii . . . . . ʒj,  
Aquam . . . . . ad ʒj.

## (48) MISTURA BUCHU ALKALINA.

℞ Potassii Nitratis . . . . . gr. v,  
Potassii Bicarbonatis . . . . . gr. xv,  
Sp. Etheris Nitrosi . . . . . ℥ xx,  
Tincturæ Nucis Vom. . . . . ℥ v,  
Tincturæ Hyoseyani . . . . . ℥ xx,  
Inf. Buchu . . . . . ad ʒj.

A valuable diuretic mixture. Also for cystitis and prostatic retention.

The Buchu infusion should be freshly prepared. The best way to administer this drug is to procure some ʒx packets of the leaves; place a packet in a pint jug previously warmed, add 1 pint of boiling water, stir, and after standing 1 hour, decant. 1 pint to be taken in 24 hours.

## (49) MISTURA CALCIS CHLORIDI.

℞ Calcis Chloridi . . . . . gr. xx,  
Tincturæ Aurantii . . . . . ʒj,  
Aquam Chlorof. . . . . ad ʒj.

Valuable for *pruritus* from any cause.<sup>1</sup> Should be given three times a day after meals in gradually increasing doses. In *hæmorrhage*, uterine or pulmonary, should be given every two to four hours.

## (50) MISTURA CAPSICI.

℞ Tincturæ Capsici . . . . . ℥ iij,  
Tincturæ Nucis Vomica . . . . . ℥ v,  
Spiritus Chloroformi . . . . . ℥ xv,  
Aquam Menthæ Piperitæ . . . . . ad ʒj.

For flatulence. An excellent "pick-me-up" for alcoholics.

<sup>1</sup> The pathology of itching and its treatment by large doses of Calcium Chloride.—*The Lancet*, August 1st, 1896.



(51) CARLSBAD MIXTURE.

R Sodii Bicarbonatis . . gr. xv,  
Sodii Chloridi . . gr. v,  
Sodii Sulphatis . . gr. xxx,  
Magnesii Sulphatis . . 3j,  
Aquam Menthæ Piperitæ ad 3j.

A morning purgative draught for plethora, obesity, gout, and rheumatic gout.

(52) MISTURA CASCARILLÆ COMPOSITA.

R Tincturæ Nucis Vomiceæ . M v,  
Tincturæ Scillæ . . M x,  
Oxymel Scillæ . . M xx,  
Infusum Cascarillæ . . ad 3j.

Chronic bronchitis and emphysema.

(53) MISTURA DIAPHORETICA.

R Spiritus Etheris Nitrosi . 3ss,  
Liq. Ammonii Acetat. . 3ij,  
Aquam Camphoræ . . ad 3j.

Diaphoretic and febrifuge.

(54) MISTURA DIGITALIS CO.

R Tincturæ Digitalis . . M v,  
Ammonii Carbonatis . . gr. iiij,  
Potassi Nitratis . . gr. v,  
Tincturæ Nucis Vomiceæ M v,  
Aquam Chloroformi . . ad 3j.

For mitral disease with failing compensation.

(55) MISTURA DIURETICA.

R Potassii Acetatis . . gr. xv,  
Spiritus Etheris Nitrosi . M xv,  
Spiritus Juniperi . . M xxx,  
Decoctum Scoparii . . ad 3j.

(56) MISTURA ETHER AMMON.

R Sp. Ammonia Aromat. . M xx,  
Spiritus Etheris . . M xx,  
Spiritus Chloroformi . . M xx,  
Aquam . . . . ad 3j.

For cardiac failure. More efficacious if accompanied by hypoderm. inj. of liq. strychn. M iiij.

(57) MISTURA EXPECTORANS.

R Ammonii Carbonatis . . M v,  
Tincturæ Scillæ . . M xv,  
Spiritus Etheris . . M xv,  
Tincturæ Strophanthi . M iiij,  
Infusum Senegæ . . ad 3j.

For acute bronchitis in the second stage.

C.M.

(58) MISTURA EXPECTORANS INFANTALIS.

R Ammonii Carbonatis . . gr. ½,  
Syrupi Scillæ . . M xx,  
Vini Ipecacuanhæ . . M iv,  
Aquam . . . . ad 3j.

For a child one year old. Given every hour in broncho-pneumonia; it may cause emesis, which is beneficial.

(59) MISTURA FERRI LAXANS.

R Ferri Sulphatis . . gr. ij,  
Magnesii Sulphatis . . 3ss,  
Acidi Sulphurici Diluti . M iiij,  
Essentiæ Menthæ Piperitæ . . M v,  
Infusum Calumbæ . . ad 3j.

(60) MISTURA FILICIS.

R Ext. Filicis Maris . . 3j,  
Syr. Zingib . . 3j,  
Tincturæ Quillaia . . 3ss,  
Aquam Chloroformi . . ad 3jss.

(61) MISTURA HYDRARGYRI BINIODIDI.

R Potassii Iodidi . . gr. v,  
Liq. Hydrarg. Perch. . 3j,  
Tincturæ Cardamomi Compositæ . . M xv,  
Aquam . . . . ad 3j.

(62) MISTURA OLEI MORRHUÆ.

R Olei Morrhua . . 3ss,  
Liquoris Calcis . . 3iiij,  
Liquor. Calcis Sacch. . M xxxvj,  
Pulv. Trag. Co. . . gr. vj,  
Olei Caryophylli . . M ½,  
Olei Cassia . . M ½.

An agreeable cod liver oil emulsion.

(63) MIST. POTAS. CITRATIS EFFERVESCENS.

No. 1.

R Potassii Bicarbonatis . . gr. xx,  
Aquam . . . . 3j.

No. 2.

Acidi Citrici . . . . gr. xv,  
Aquam . . . . 3ss.

An agreeable effervescing vehicle for quinine and other drugs.

## (64) MISTURA RICINI CALCIS.

℞ Olei Ricini . . .	℥ss,
Liquoris Calcis . . .	℥ss,
Tincturæ Quillaie . . .	℥ xv,
Syrupi . . .	℥ xx,
Olei Menthæ Piperitæ . . .	℥ j.

℥j every hour for diarrhœa and unhealthy stools in children.

(65) MISTURA SEDATIVA  
INFANTALIS.

℞ Ammonii Bromid . . .	gr. iv,
Tincturæ Belladonnæ . . .	℥ iv,
Glycerini . . .	℥ x,
Aquam . . .	ad ℥j.

A harmless sedative for a child of one year old.

## (66) MISTURA STOMACHICA.

℞ Magnesii Carbonatis . . .	gr. x,
Sodii Bicarbonatis . . .	gr. xv,
Acidi Carbolici pur. . .	℥ j,
Tincturæ Rhei Compositæ . . .	℥ xv,
Infusum Calumbæ . . .	ad ℥j.

Tea-drinker's dyspepsia and pyrosis.

## (67) MISTURA STRYCHNINÆ.

℞ Liq. Strych. Hydrochlor. . .	℥ iiij,
Acidi Nitro-Hydrochlorici Diluti . . .	℥ v,
Tincturæ Capsici . . .	℥ j,
Tincturæ Lavand. Co. . .	℥ v,
Aquam . . .	ad ℥j.

The tonic for old age.

## (68) MISTURA PRO TUSSI.

℞ Tincturæ Camph. Co. . .	℥ x,
Vini Ipecacuanhæ . . .	℥ v,
Oxymellis Scillæ . . .	℥j,
Aquam Anisi . . .	ad ℥j.

For chronic bronchitis.

## (70) NEBULA ALKALINA.

℞ Sodii Bicarbonatis . . .	gr. xij,
Boracis . . .	gr. xij,
Acidi Carbolici . . .	gr. iv,
Glycerini . . .	℥ xl,
Aquam . . .	ad ℥j.

To be sprayed into the anterior nares for ozæna and ulceration.

## (72) NEBULA ZINCI CHLORIDI.

℞ Zinci Chloridi . . .	gr. x,
Acidi Hydrochlorici Diluti . . .	℥ j,
Aquam Destillatam . . .	℥j.

Sprayed into the anterior nares for chronic rhinitis; may be also used as a throat spray for chronic laryngitis. gr. xx—℥j cuts short an attack of acute laryngitis, and should be used as follows: pull the tongue well forward and spray interior of larynx; apply cold compress to throat at night, and in the morning let patient inhale chloroformi ℥ xx, Tr. Benzoin Co. ad ℥j in a pint of boiling water every few hours. (See Vapores below.)

(75) PASTA<sup>1</sup> RESORCIN COMP.

℞ Resorcin . . .	℥j.
Zinci Oxidi . . .	℥ij.
Pulv. Amyli . . .	℥ij.
Petrol. Moll. . .	℥j.

Mix well. A valuable protective paste. For chronic conditions, Salicylic or Carbolic Acid (20—60 grs.) may be added.

## (76) PASTA SULPHURIS.

℞ Sulph. Precipit. . .	℥j.
Zinci Oxidi . . .	℥j.
Silicious earth . . .	℥ss,
Adep. Benz. . .	℥j.

A protective paste for chronic skin diseases.

## (77) PIGMENTUM ACIDI CHROMICI.

℞ Acidi Chromici . . .	gr. 50,
Aquam Destillatam . . .	ad ℥j.

To paint unhealthy ulcers.

## (78) PIGMENTUM ACIDI SALICYLICI.

℞ Acidi Salicylici . . .	℥j,
Extracti Cannabis Indicæ . . .	gr. viij,
Collodii Flexilis . . .	℥vj,
Ætheris . . .	℥ij.

Paint on every night to remove corns.

<sup>1</sup> Pastes are stiff ointments which act as protectives and drying agents.

(79) PIGMENTUM CADIS.

R Ol. Cadi. . . . . 3j,  
 Spiritus Vini Meth. . . . . 3j,  
 Sapo Mollis . . . . . 5ij.  
 For psoriasis of scalp and lupus erythematosus.

(80) PIGMENTUM CREASOTI.

R Creasoti . . . . . M ij,  
 Acidi Salicylici . . . . . gr. xx,  
 Collodi . . . . . 3j.  
 Very efficacious for lupus erythematosus.

(81) PIGMENTUM IODI COMPOSITUM.

R Iodi . . . . . 3j, } By  
 Acidi Carbol. Liq. . 3iv, } weight.  
 Rub together in a warm mortar and dissolve, warming if necessary. Resembles Costor's Paste. For intractable ringworm; apply once a week to the scalp with a hog's bristle brush.

(82) PLASTER MULLS (Emplastra), with composition as follows, are obtainable at most large pharmaceutical chemists.

- (a) Empl. Ac. Salicylic (5 to 15 %).
- (b) Empl. Ac. Salicyl. (5%), and Ac. Carbol. (10 %).
- (c) Empl. Ac. Salicyl. (10 to 50 %), and Creosote (20 to 50 %).
- (d) Empl. Hydrarg. (10 to 30 %).
- (e) Empl. Hydrarg. (20 %), and Ac. Carbol. (7½ %).
- (f) Empl. Hydronaphthol (5 and 10 %).
- (g) Empl. Ichthyol (5 and 10 %).
- (h) Empl. Ichthyol (10 %), and Chrysorobin (10 %).
- (i) Empl. Resorcin (10 and 15 %).
- (j) Empl. Zinci Ox. (20 %), and Sulphur (1 %).
- (k) Empl. Zinci Ox. (10 %), and Ac. Salicyl. (5 %).
- (l) Empl. Zinci Ox. (10 %), and Ichthyol (5 %).

(83) PILULA ASAFÆTIDÆ CUM VALERIANA.

R Asafœtidæ . . . . . gr. iij,  
 Zinci Valerianatis . . . . . gr. j.  
 Flatulence, "nervousness," and the manifestations of hysteria generally.

(84) PILULA DIGITALIS COMPOSITA.

R Pulveris Digitalis . . . . . gr. j,  
 Pulveris Scillæ . . . . . gr. j,  
 Pilulæ Hydrargyri . . . . . gr. j.

Valuable in cardiac dropsy, and as a diuretic in ascites. It is apt to salivate unless the bowels are acting regularly.

(85) PILULA ELATERINI.

R Pulveris Elaterini Compositi . . . . . gr. ij,  
 Extracti Hyoscyami . . . . . gr. ¼.  
 ½ gr. Elaterini in each pill. Diuretic and drastic pill for ascites.

(86) PILULA FERRI ALKALINA (Blaud).

R Ferri Sulphatis . . . . . 5j,  
 Potassii Carbonatis . . . . . 3j,  
 Pulveris Tragacanthæ . . . . . gr. xij,  
 Glycerini . . . . . M x.

Misce bene; fiant pilulæ, xxiv. Should be freshly prepared. Two a day, increased to eight, after meals.

(87) PILULA FERRI COMP.

R Ferri Sulph.  
 Zinci Valerian . . . . . āā gr. j,  
 Ext. Aloes Barbadianis  
 Ext. Nucis Vomicae . . . . . āā gr. ¼.

For the amenorrhœa and anæmia of hysterical girls.

(88) HALFORD'S GOUT PILL.

R Extracti Colchici Acetici gr. ½,  
 Pulveris Ipecacuanhæ  
 Compositi . . . . . gr. j,  
 Extracti Colocynthidis  
 Compositi . . . . . gr. j.

Twice or thrice a day for acute gout.

(89) PILULA HYDRARGYRI COMPOSITA.

R Pilulæ Hydrargyri . . . . . gr. j,  
 Pulveris Ipecacuanhæ . . . . . gr. ½,  
 Pilulæ Rhei Compositæ . . . . . gr. ij.

May be given every night to reduce arterial tension.

(90) PILULA PODOPHYLLI  
COMPOSITA.

℞ Resinæ Podophylli . gr. j,  
Pulveris Ipecacuanhæ . gr. j,  
Hydrargyri Subchloridi . gr. j,  
Extracti Hyoseyami . gr. ij.

A useful liver pill in hepatic congestion.

## (95) PULVIS ALTERATIVUS.

℞ Hydrargyri cum Cretâ . gr. j,  
Pulveris Cinnamomi  
Compositi . . gr. j,  
Pulveris Rhei . . gr. ij,  
Magnesii Carbonatis . gr. ij.

Dose for a child one or two years old.

## (96) PULVIS GUAIACI COMPOSITA.

℞ Pulveris Guaiaci Resinæ  
Sulphuris Precipitati  
Magnesii Carbonatis . āā gr. xx.  
3j to iij every evening. A valuable  
alterative for gout and rheumatism; and  
for sciatica (gr. xx, t. d.).

## (97) PULVIS MIRABILIS.

℞ Bismuth. Carbonatis . gr. v,  
Sodii Bicarbonatis . . gr. v,  
Pulv. Rhei . . gr. j,  
Pulv. Nucis Vomicae . gr. ½,  
Pulv. Cinnamon. Co. . gr. 1½.

To be taken before meals. For  
dyspepsia.

(98) PULVIS SCAMMONII CUM  
HYDRARGYRO.

℞ Pulveris Scammonii . gr. iij,  
Hydrargyri Subchloridi . gr. j,  
Pulveris Zingiberis . gr. ij.

Dose for a child two years old. An  
aperient powder for thread-worms.

## (100) TABLOIDS.

One of the chief objections to medicines  
in the form of tabloids appears to be  
their insolubility and the possibility of  
their not passing into the stomach until  
some considerable time after they are  
swallowed. Dr. J. S. Bristowe (Clin.  
Lects. and Essays on Diseases of the  
Nervous System) narrates a case in  
which small tabloids containing morphia  
were found in the folds of the œsophagus  
after death, and I have met with similar  
cases. A draught of water or some food  
should always follow their administra-  
tion.

(101) UNGUENTUM CALAM. CUM  
OLEO.

℞ Calaminæ  
Zinci Oxidi . . . āā gr. x,  
Olei Lini . . . 3ss,  
Adeps Benz . . . 3j.

A soothing ointment. Useful for  
pruritus.

## (102) UNGUENTUM CREOLINI COMP.

℞ Creolin . . . 3j,  
Unguenti Hyd. Ammon. 5iij,  
Sapo Mollis . . . 3j,  
Pet. Mollis . . . ad 3j.

For psoriasis or ch. eczema (in certain  
stages).

## (103) UNGUENTUM METALLORUM.

℞ Unguenti Hydrargyri .  
Unguenti Plumbi Car-  
bonatis . . .  
Unguenti Zinci Benzoati āā 3 iv.

For irritable or acute eczema.

(104) UNGUENTUM PETROLATI  
COMP.

℞ Hydrargyri Ammoniati . gr. x,  
Liquoris Carbonis Deter-  
gentis . . . 3ss,  
Paraffini Mollis . . . 3j.

A mild tar and mercury ointment,  
useful in many skin diseases.

(105) UNGUENTUM SALICYLICI ET  
CARBOLICI.

℞ Ac. Salicylici  
Ac. Carbolic . . . āā gr. xxx,  
Vaselin . . . ad 3j.

Stimulating ointment for chronic skin  
affections.

## (106) UNGUENTUM SULPHURIS CO.

℞ Sulph. Sublimat. . . gr. xxx,  
Acid. Carbol. . . ℥ viij,  
Sapo Mollis . . . 3ss,  
Adipis Benz. . . ad 3j.

For acne. Should be rubbed in night  
and morning. In obstinate cases Sapo  
Mollis 5ij—5iij and more sulphur may be  
added.



(110) VAPORES (Inhalations).

*Directions.*—A teaspoonful to be added to a pint of boiling water, to be inhaled for five minutes every night and morning from a narrow-necked jug or suitable inhaler. In this way use Tr. Benzoini Co. as an expectorant and local sedative in bronchitis and laryngitis; Tr. Iodi as a stimulant in chronic catarrh. Ext. Lupuli will allay irritability of mucous membrane. Ol. Eucalypti, Terebene, Creosote Ol. Pini Sylvestris, may all be employed in the same way (strength  $\mathfrak{m}$  40—Oj), and certainly produce alterative effects in chronic catarrh if persevered with for several weeks.

If intended for Eustachian medication the following directions should be observed:—About six times in the five minutes well fill the mouth with steam, close the nostrils with the thumb and forefinger, shut the mouth and expire forcibly so as to drive the vapour towards the ears.

(111) VAPOR CARBONIS (Anti-Catarrhal Smelling-Salts).

℞ Acidi Carbolici . . . gr. xxx,  
Ammonii Carbonatis . . .  $\mathfrak{z}$ j,  
Carbonis Pulveris . . .  $\mathfrak{z}$ j,  
Olei Lavandulæ . . .  $\mathfrak{m}$  xx,  
Tincturæ Benzoini Com-  
positæ . . .  $\mathfrak{z}$ ss.

A dry inhalation for catarrh. Boracic acid, finely powdered, may be used as a snuff.

(112) VAPOR SICCUS.

℞ Olei Pini Sylvestris . . .  $\mathfrak{z}$ j,  
Olei Eucalypti . . .  $\mathfrak{z}$ j,  
Tincturæ Benzoini Com-  
positæ . . .  $\mathfrak{z}$ ij,  
Creasotum . . . ad  $\mathfrak{z}$ j.

Ten or fifteen drops to be placed on the sponge of an oro-nasal inhaler previous to use.

(112) RESISTANCE EXERCISES.

(Referred to in the treatment of heart disease, p. 110.)

These exercises, as practised at Nauheim, Hesse-Nassau, Germany, comprise a series of movements, each, as it is being performed, being gently resisted by the nurse or operator. They are never to be repeated twice in succession. The patient, while performing them, should be carefully observed, and if any symptom of interference with the circulation or respiration (*e.g.*, breathlessness, change of tint about the lips or cheeks, dilatation of nostrils, contraction of corners of mouth, moisture on the forehead, yawning) appear, the movement must be at once stopped and a rest taken. During the movements the patient should breathe regularly, and it may be advisable that he should count in whispers so as to achieve this end. The attire should be perfectly free.

The resistance is accomplished by the operator placing the palmar surface of his hand on the aspect of the patient's limb towards which the movement is directed or, in the case of body movements, on that aspect towards which the movement is being carried out. While undergoing the course of exercises, the diet must be liberal, but little importance is attached to the nature of the food, so long as it is plain and nutritious. The movements must always be followed by a period of rest.

The movements must always be begun very cautiously, *i.e.*, the simpler ones first; and with every care to avoid even the appearance of fatigue. Each exercise can be varied considerably by the operator (1) by modifying the *degree of resistance*, and (2) the *speed*—the slower the movement the greater the strain.<sup>1</sup>

a. Resistance exercises, as carried out by the Drs. Schott, consist of a series of 19 movements given in an anatomical series, from which the physician, after studying them

<sup>1</sup> A number of nurses of both sexes have now been trained by Dr. J. Fletcher Little to carry out this treatment in England, and also to administer the baths, the ingredients of which can be obtained from Buchner, 149, Houndsditch, E.C.

carefully, can select and arrange a certain number according to the nature and progress of the case from day to day. All, excepting 6 to 8 and 12 and 14, can be done with the patient in the recumbent position, though not to the fullest extent.

(1) The arms are stretched out in front of the body at the level of the shoulders with the palms meeting each other. The arms are then carried outwards laterally in line with each other, and thereafter are brought back to their first position.

(2) The arm and hand are placed in the fully supinated position, hanging down, and the forearm is flexed upon the arm, without any movement of the latter, until the fingers touch the shoulder; thereafter the arm is extended to its original position. This movement is carried out first with one arm and then with the other.

(3) The arms, hanging down, are supinated and raised outwards until the thumbs meet over the head, after which they are brought back to their original position.

(4) The fingers of the hands, flexed at the first phalangeal joints, are pressed together in front of the lowest part of the body, and the arms are raised until the hands are above the head, after which they are brought back to their original position.

(5) The arms, hanging in the position of "attention," are raised forwards parallel to each other until they are elevated to a vertical position, and are then brought back to the position from which they started.

(6) The body is bent forwards and then brought back to the erect position, the knees not being moved. (Enquiry should be made of the patient for any sensation in the head, and if such is present, the exercise must be stopped.)

(7) The body is rotated without any movement of its feet, first to one side, then to the other, and finally back to its original position.

(8) The body is bent laterally as far as possible, first to one side, then to the other, and afterwards restored to its original erect posture.

(9) This is a movement precisely similar to No. (1), except that it is carried out with the fists clenched.

(10) The arms are moved in the same way as in exercise No. (2), but the fists are firmly clenched.

(11) The arms, starting from the position of "attention," describe a circle by moving forwards and upwards until they are raised vertically. Each palm is then turned outwards and the arms descend backwards to their original position.

(12) The arms, starting from the position of "attention," are moved upwards and stretched backwards as far as can be done without bending the trunk, and are then brought back to their original position. (For this movement the patient should be facing a looking-glass for the attendant to watch his face.)

(13) The patient, standing with the feet side by side and supporting himself by leaning with one hand upon any object, flexes the opposite thigh as far as it is possible, and afterwards extends it until the feet are again side by side. Thereafter, leaning upon the other hand, he carries out a similar movement with the other thigh.

(14) The patient, leaning as in the last exercise, first bends the whole lower extremity of one side, kept extended, as far forwards as possible, then backwards as far as he can, and afterwards brings it beside the other. A similar movement thereafter is carried out with the other leg.

(15) The patient, leaning both hands in front on the back of a chair, the patient flexes first one leg and then the other upon the thigh as far as he can.

(16) Resting on one hand, the patient raises the extended opposite lower extremity outwards as far as possible, and then brings it beside its fellow. A similar movement is then carried out with the other limb.

(17) The arms, held horizontally outwards, are rotated forwards and backwards at the shoulder joint, the operator clasping the patient's wrists and resisting.

(18) The hands, held in the extended position, are first bent backwards and then forwards as far as possible, after which they are brought back to their original position.

(19) The feet, held in their ordinary position, are first bent downwards and then upwards as far as possible, after which they are brought back to their original position.

b. Resistance exercises, as given in Nauheim by Dr. Grödel, and arranged by him in progressive order, beginning with the simplest and least tiring movements.

GROUP I.—1. Flexion and extension of the fingers.

2. Flexion and extension of the hand.

3. Flexion and extension of the feet.

4. Flexion and extension of the elbow.

GROUP II.—1. Pronation and supination of the arm, with the arm fully extended.

2. Abduction and adduction of the arms (to shoulder level only).

3. Flexion and extension of the knees.

4. Arms raised in front of the body and returned (to shoulder level only).

GROUP III.—1. Arms, held horizontally, are brought forwards and backwards.

2. The lower extremities, fully extended, are turned outwards and inwards (sitting).

3. Complete abduction and adduction of the arms (the hands meeting above the head).

4. Abduction and adduction of the legs (sitting).

GROUP IV.—1. Arms extended, raised in front of body and up above head, then returned.

2. Flexion and extension of hip, with flexed knee (sitting or lying).

GROUP IV.—*continued.*

3. Arm hanging at side of body is moved backwards and forwards like the pendulum of a clock.

4. Flexion and extension of the trunk.

GROUP V.—1. Arms extended and fists clenched ; arms raised in front of body to shoulder level, and brought back.

2. Flexion and extension of thigh, patient standing, and supporting himself on a chair with opposite hand.

3. Flexion and extension of the head.

4. Rotation of the trunk.

GROUP VI.—1. Flexion and extension of the hip, with extended knee (sitting or lying).

2. Rotation of the head.

3. Trunk bent sideways and back.

4. Abduction and adduction of the extended lower extremities, patient standing and supported by a chair.

GROUP VII.—1. Sawing—the arm being held as in the act of sawing, is moved forwards and backwards and returned to original position.

2. The lower extremities moved forwards and backwards like the pendulum of a clock (patient standing).

3. Raising of the trunk (lying down).

4. Flexion and extension of the hip (lying down).





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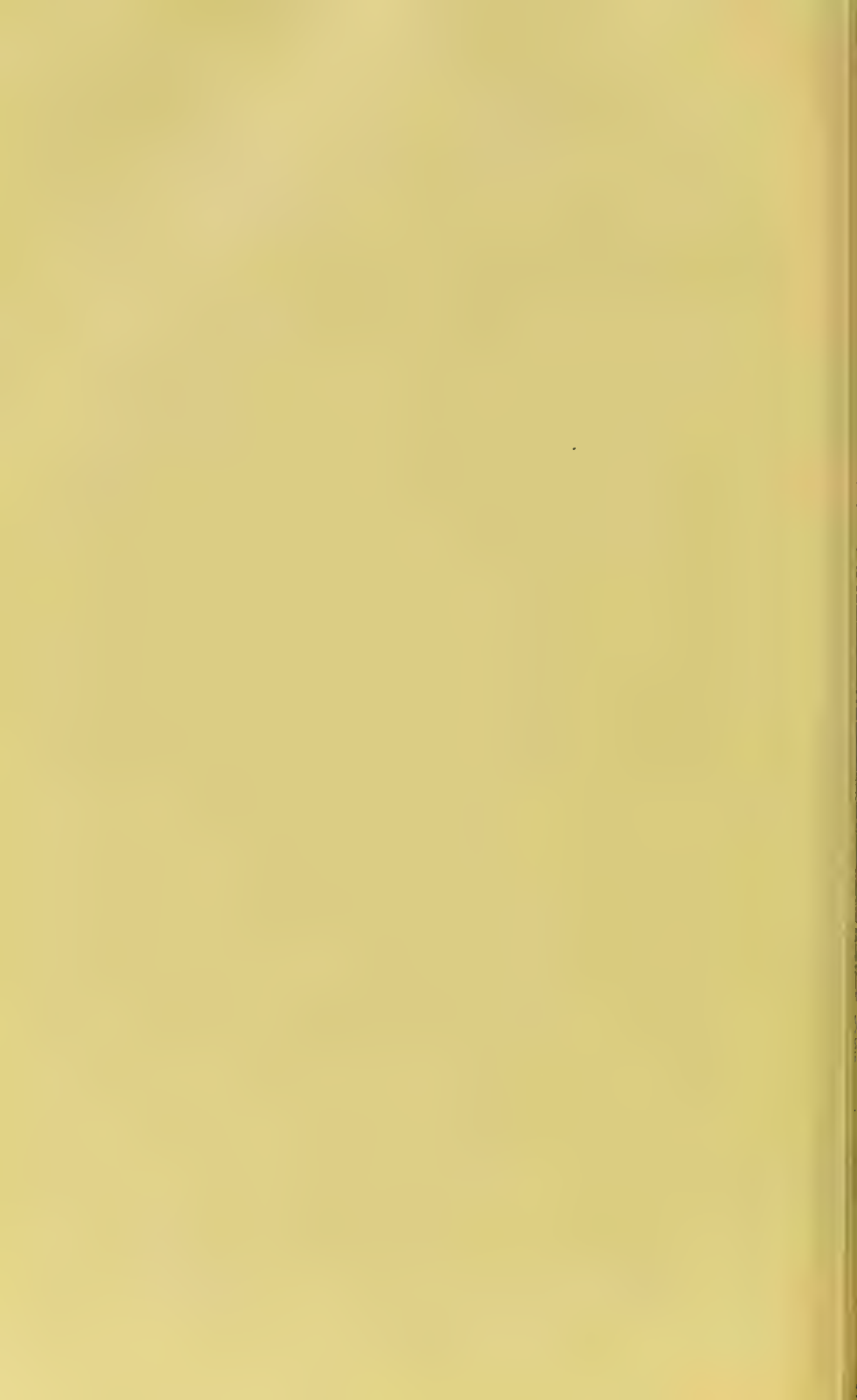


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